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A.M.A. ARCHIVES OF OTOLARYNGOLOGY

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Arthur J. Cracovaner

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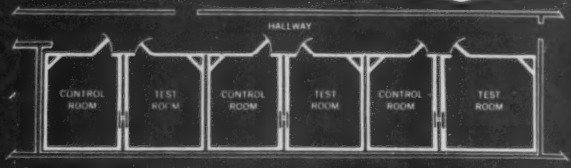

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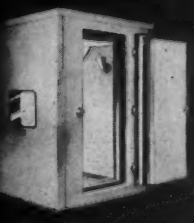

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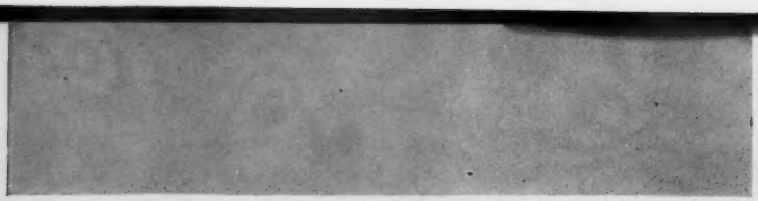

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A.M.A. ARCHIVES OF
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Hyperkeratosis of the Larynx

ARTHUR J. CRACOVANER, M.D., New York

Hyperkeratosis of the larynx is a very important and perplexing subject to the laryngologist. It is considered a precancerous lesion, and in many cases such growths actually do metamorphose into true cancers. However, many of the lesions are benign, and so we feel that radical treatment is frequently unnecessary. The problem is, how do we differentiate the benign lesion from those that may become malignant and those already showing evidence of early malignancy? The next question is, can we formulate methods of handling these cases that will eliminate the disease and at the same time avoid radical and mutilating procedures? At the outset it may seem that the problem is not a very difficult one, and yet the experienced laryngologist frequently finds himself confronted with instances that are baffling as to the course of treatment that should be followed.

Hyperkeratotic areas in the larynx may develop characteristic malignant changes without visual evidence. Only on microscopic examination is one able to reveal the presence of malignant changes. Therefore, a keratotic lesion must always be considered potentially malignant until microscopic examination is performed. It must be managed with the idea that at any time it may become malignant.

Submitted for publication Jan. 27, 1959.

Presented at a meeting of the International College of Surgeons, Southeastern Regional Assembly, Jan. 4-7, 1959, Miami Beach, Fla.

From the Department of Bronchoesophagology and Laryngeal Surgery, New York Eye and Ear Infirmary, and the Department of Otolaryngology, Lenox Hill Hospital.

Clinical Appearance

On clinical examination of the larynx, a hyperkeratotic growth is a raised, dense, thickened area of the superficial layer of the epithelium. When it is entirely or partially covered with white superficial plaques and there is evidence of inflammation it is called leukoplakia. It may be localized or widespread; it may be single or multiple. The lesion may involve one or both cords. It is sometimes grayish white, and at other times there is a red thickening of the cord. Keratosis may appear as a white or gray warty papillomatous lesion, and sometimes it may have a sharp spiny irregular surface. The growth may spread to the ventricle or subglottic area or both and become large enough to cause laryngeal obstruction.

Microscopic Appearance

On microscopic examination, hyperkeratosis is seen as a thickening of the epithelium, especially the cornified stratum. In one form, the superficial layers are markedly thickened, while the deeper layers are not so greatly involved. In some of these, there is no inflammatory reaction in the underlying connective tissue and no cell atypism is to be seen. In leukoplakia, however, there is evidence of chronic inflammation in the mucosal layers and submucosa, as evidenced by the presence of lymphocytes and plasma cells. In addition, atypical

cells may be seen in the deeper layers and in the prickle-cell and basal-cell areas, but the basal layer remains unbroken. These atypical cells are indicative of the possibility of further metamorphosis into a malignant growth. The atypical cells may increase in number and gradually show characteristics of carcinoma, that is, atypical chromatin arrangements, large nuclei and nucleoli, hyperchromatism, and large, atypical-shaped squamous cells. With sufficient changes of this type in the cells we are dealing with carcinoma in situ.

Keratotic lesions showing chronic inflammation and involvement of the deeper layers of epithelium are more likely to metamorphose into malignant growths. The presence of atypical cells is considered the beginning of carcinomatous changes. If these changes are pronounced enough, the lesion is considered carcinoma in situ. Infiltration of these cells beyond the basal layer into the subepithelial tissue indicates the presence of true invasive carcinoma.

The microscopic picture, then, is essential in determining the type of lesion we are dealing with. Either a biopsy specimen or, if the growth is small, the entire specimen must be investigated microscopically. We should keep in mind that a biopsy specimen may be taken from an area that as yet does not show malignant changes. Also, these malignant changes are slow in developing and may not be evident at the time the biopsy specimen is obtained. Specimens from several areas of a large tumor may overcome the possibility of missing the malignant areas.

It is important then to consider carefully these cases of keratosis of the larynx that reveal atypical cells on microscopic examination. At the same time it is not always necessary to be too radical in the handling of these growths. It would seem that each case must be given special attention and be judged according to the extent of the lesion, the size, the appearance, and the site, as well as the microscopic picture. There are so many variations that it is difficult to assign definite rules for treatment of these conditions, but I plan to present certain typical cases and indicate how I feel they are best managed.

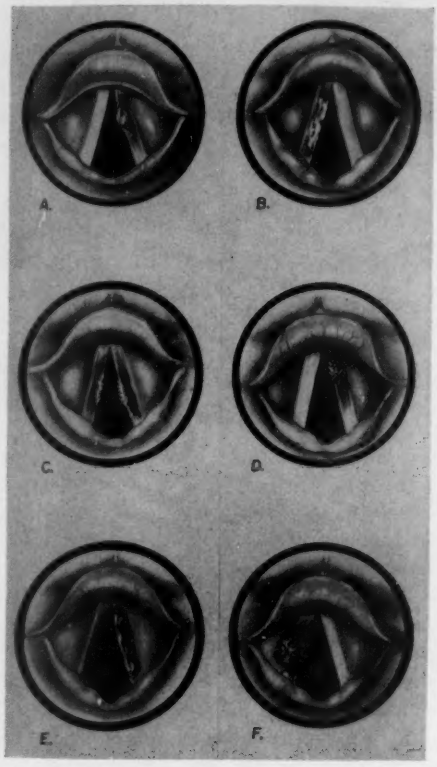

Fig. 1.—Early hyperkeratoses: *A*, red thickening, *B*, white plaques, *C*, white, brittle appearance, *D*, papillomatous, *E*, leukoplakia (with inflammation), and *F*, keratosis (papillary type, possibly malignant).

The early hyperkeratotic lesion may appear as a small, red thickening on the edge of a vocal cord (Fig. 1*A*). It may be in the anterior or middle third of the cord or in both areas. Hyperkeratotic lesions in general are thought to be due to chronic irritation. The irritation may come from overindulgence in alcohol, from excessive smoking, from abuse of the voice, and from inhalation of dust and fumes. Repeated and chronic infection in the sinuses or pharynx may be a source of chronic infection that affects the larynx. It would be wise, then, to eliminate these sources of

irritation and infection and to observe the effect on the larynx if the lesion is a small one. If it does not disappear, then a direct laryngoscopy is done and the thickened area is removed. The patient should be observed, and if the lesion recurs it is again removed.

The keratotic growth may appear as a white or grayish white elevated thick patch on the upper surface and on the edge of the vocal cord or on both vocal cords (Fig. 1*B*). The cords themselves will show evidence of chronic inflammation. On the other hand, a keratosis may be seen as a white, warty, brittle-appearing excrescence on one or both cords (Fig. 1*C*). Other hyperkeratotic lesions appear as white or red papillary growths (Fig. 1*D*).

Some of these lesions may subside if the patient is placed on a regimen to eliminate chronic irritation and infection. Usually, however, it is necessary to strip the involved area from the vocal cord by direct laryngoscopy and then insist on the elimination of the causative irritating and infectious factors.

The patient must be kept under close observation for the possibility of recurrence. The lesions that are surrounded by inflammation (Fig. 1*E*) and the warty, papillomatous growths (Fig. 1*F*) seem more likely to become malignant, and yet the sessile, red thickening on a vocal cord is sometimes reported as carcinoma in situ (Fig. 2*G*).

If the keratotic lesion has a tendency to recur after repeated removals, then more radical means must be undertaken. We prefer to remove the involved cord by laryngofissure. It is interesting to note that between recurrences the intervals tend to become shorter and the tumor seems to grow faster. We are inclined to remove by laryngofissure the warty papillomatous lesion on an inflamed vocal cord (Figs. 1*F* and 2*H*), because this type has a tendency to grow large quickly and to spread to the ventricle and the subglottic areas, and it is difficult to remove them entirely by direct laryngoscopy.

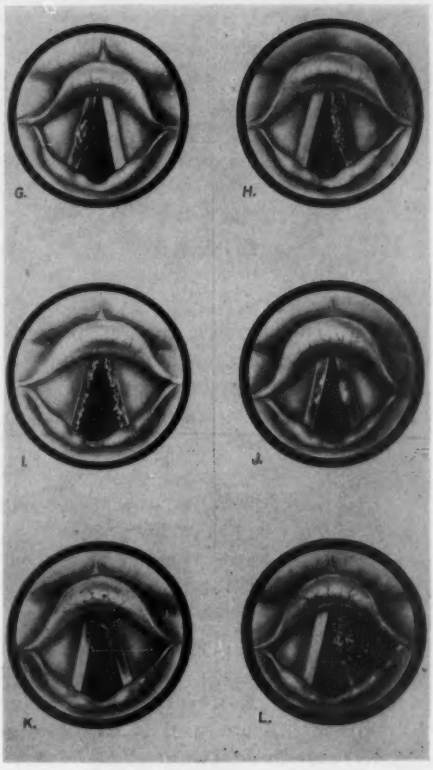

Fig. 2.—*G*, Carcinoma-in situ, *H*, keratosis (papillary type, possibly malignant), *I*, and *J*, bilateral hyperkeratosis, *K*, keratosis involving anterior commissure, and *L*, extensive lesion, usually malignant.

If any of the growths already mentioned show on microscopic examination the presence or suspicion of malignant change, the growth is treated as a carcinoma and removal is done by laryngofissure. The presence of a few atypical cells may alert us to the fact that the patient should be closely observed and, any sign of recurrence would call for operative removal of the vocal cord.

The treatment of keratotic lesions involving *both vocal cords* is more serious and perplexing (Fig. 2*I*). Some of these growths may show evidence of beginning malignant changes. The lesion may extend slightly on to the ventricle or the subglottic area; yet it may be superficial, and both cords may be mobile. On one vocal cord there may be white plaques and on the other a red thickening (Fig. 2*J*). The only sur-

gical approach to these cases would be a laryngectomy. It seems to me that this treatment is too radical. Before subjecting these patients to laryngectomy, I prefer to recommend radiation therapy. Some of these cases do well with such treatment, but others must be closely observed for recurrences. Then a laryngectomy becomes necessary.

The question of radiation dosage has been raised. Some feel it is sufficient to give partial tumor doses, being guided by the progress and the effectiveness of the treatment. But others insist that it is important to administer the full tumor dose. It is claimed that the vocal cords return to normal in appearance, but I find that with full dosage the vocal cords remain thickened and white. This is not important if one can obtain a cure. Even though the reactions to the tumor doses are severer, I would rather not depend on partial radiation therapy.

I have been asked why I do not recommend radiation therapy when one cord is involved in hyperkeratosis, with or without beginning malignant changes. I believe that surgery is a more certain method of curing these conditions, and I would like to see the entire lesion removed when it is limited to one cord. Besides, the morbidity in radiation therapy is much greater and longer than in a laryngofissure, but if eradicating the disease requires as mutilating an operation as laryngectomy, I prefer to try radiation therapy first. Of course, if the lesion is definitely malignant or shows a tendency to invasive cancer, then a laryngectomy is performed.

We have encountered a few cases where one cord is almost entirely involved and the lesion extends across the anterior commissure onto a small portion of the other cord (Fig. 2*K*). A bilateral thyrotomy (Kemler) and an anterior partial laryngectomy (Jackson) are relatively conservative operative measures that can be performed in such cases.

Occasionally we have seen cases where the tumor is a large, papillomatous growth

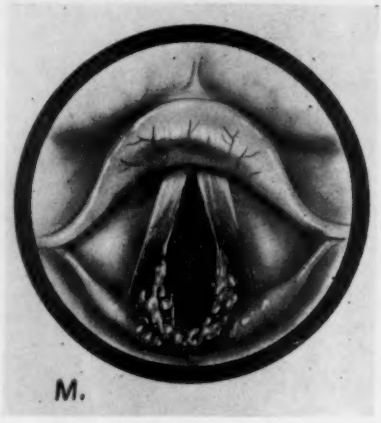

Fig. 3.—*M*, Pachydermia.

that not only involves one vocal cord or part of both cords but also extends subglottically or extends to the ventricle and even to the epiglottis and aryepiglottic fold (Fig. 2*L*). The tumor looks malignant, and yet many biopsy specimens are reported as hyperkeratotic, perhaps with atypical cells. It is very possible that biopsies are done in areas that have not as yet turned malignant. These cases are best treated as true invasive cancers, and laryngectomy should be done. Usually, on section of the larynx after removal, the presence of cancer is definitely established.

There is a type of diffuse hyperkeratosis that involves the interarytenoid area and a portion of each vocal cord posteriorly. It appears as a dry, wrinkled, irregular, grayish-white or inflamed thickening of the mucous membrane (Fig. 3*M*). This type of hyperkeratosis of the larynx is sometimes called pachydermia. The treatment of these cases is difficult because they have a tendency to recur, but only rarely does a case become malignant. It is best to strip the involved area under direct laryngoscopy, to eliminate any cause of irritation or infection, and to observe the patient at frequent intervals. If malignant changes occur a laryngectomy must be done. Radiation does not seem to help these cases.

Conclusions

Hyperkeratosis of the larynx is a precancerous lesion and must be treated as a potential malignancy.

Small localized lesions are treated by stripping the cords under direct laryngoscopy. Irritation due to alcohol, tobacco, abuse of the voice, infection, etc. are eliminated.

Lesions that recur on one vocal cord or a papillomatous-appearing lesion on one vocal cord are removed by laryngofissure.

Lesions that recur on both vocal cords are treated by radiation. If this is unsuccessful a laryngectomy is performed.

Extensive involvement of the larynx by a warty papillomatous-appearing lesion is treated by laryngectomy.

Dr. George K. Higgins helped in preparing the pathological aspects of this paper.

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Bilateral Eosinophilic Granulomas of the Mastoid Processes

Report of a Case in a Two-Year-Old Boy, with Review

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In 1952, Harris and Spencer [1] reported a case of a 3-year-old girl who suffered from eosinophilic granulomas of both mastoids, and they stated that, until then, only three instances of bilateral xanthomatous lesions of the mastoids had been described since the original description, by Schüller, of the complex known as Hand-Schüller-Christian syndrome.[2-4]

Therefore, in view of the extreme rarity of this distribution of lesions, which are in themselves sufficiently rare, we feel that it is worth while to report the following case.

Report of Case

A 2-year-old white boy was admitted to the Charles V. Chapin Hospital on June 28, 1957, complaining of a painful swelling behind his left ear of two days' duration.

Since his fourth month the child had had frequent upper respiratory infections, and eight weeks prior to his admission he complained of fever and left-sided earache, which was treated by his family doctor. The fever subsided, but four weeks later the fever reappeared and subsided again. Two weeks prior to his admission his mother noticed a swelling behind his left ear. X-ray studies showed a large radiolucent defect in the left mastoid process, and he was admitted to the hospital.

The maternal grandmother, an aunt, and an uncle were said to suffer from pulmonary tuberculosis, although intimate contact with this patient was denied.

Physical Examination.—Temperature 100.4 F, pulse 120, respirations 24. The patient was a well-developed, well-nourished, irritable, white child in no acute distress. The head appeared large, with frontal bossing. The eyes presented a conspicuous internal strabismus, the left more pronounced than the right. Examination of his ears showed edema and bulging into the right external canal and a slightly thickened and bulging right drumhead. The left external auditory canal was very swollen, suggesting a posterior wall furuncle. The left drumhead was visualized with difficulty and was thickened and tense. Over the left mastoid process there was a hard, slightly tender swelling, not red or fluctuant.

The remainder of the physical examination was essentially negative.

Laboratory Findings.—Hemoglobin 10.4 gm. (74%), white cell count 11,000. The differential white cell count was normal, and the patch test was negative.

Culture of the right ear disclosed Pseudomonas aeruginosa; that of the left ear yielded Staphylococcus pyogenes var. albus.

Urinalysis was negative.

The blood serology was negative.

X-Ray.—There was a large oval radiolucent defect, having a relatively smooth margin and measuring about 2.5 cm. in diameter, in the posterior portion of the left mastoid process (Fig. 1). Films of the chest and a skeletal survey were negative.

Operation.—On the 11th hospital day surgical exploration of the left mastoid was undertaken, and a large gray firm mass was removed from the area.

Fig. 1.—A well-defined area of radiolucency is seen, involving the left mastoid process.

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From the Departments of Otorhinolaryngology, Pathology, and Radiology, of the Roger Williams General Hospital and Charles V. Chapin Hospital.

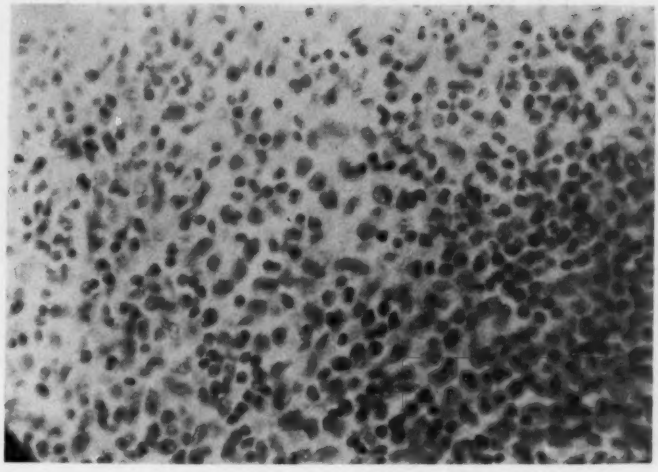

Fig. 2.—Mass from left mastoid process. The large pale cells are of reticuloendothelial origin; the small darker ones are eosinophils.

Frozen section showed sheets of pale cells as well as myriads of eosinophils scattered diffusely and focally throughout the section. A presumptive diagnosis of eosinophilic granuloma was made.

Pathologic Examination.—The specimen consisted of an irregularly shaped, lobulated, greenish-brown to yellow piece of tissue measuring 3×2×1 cm. It was very mushy and soft but sectioned easily, revealing a greenish-yellow-brown cut surface. The tissue was embedded in toto.

Microscopic Findings: There were large masses and sheets of large, pale, pleomorphic cells, regarded as histiocytic in origin. They possessed for the most part, a single, round, ovoid, lobulated, or notched nucleus, although a few contained two or even more nuclei. There was a scanty to moderate cytoplasm. Mitoses were occasionally seen, and a few instances of cytophagocytosis were noted.

Alternating with or intimately admixed with these large pale cells were large masses of eosinophils. In some areas myriads of these cells imparted a brilliant red color to the microscopic field. Most of the eosinophils were polymorphonuclear cells, but some appeared to be eosinophilic myelocytes (Fig. 2).

In a few areas fibrosis was the predominant feature, with little cellular infiltration or none at all.

Occasional small foci of lymphocytes were present, and small areas of necrosis were seen.

Pathologic Diagnosis. — Eosinophilic granuloma of bone.

Treatment.—Six x-ray treatments of 100 r each were administered to the area.

The patient was discharged on his 34th hospital day as improved, in the care of his family physician.

The child was readmitted, on Feb. 13, 1958, to the Roger Williams General Hospital, complaining of a swelling behind the right ear of two months duration. There was no earache, and the mastoid area was tender.

Physical Examination.—There was a tender swelling behind the right ear the size of a walnut. The remainder of the physical examination was noncontributory.

Laboratory Findings.—Hgb. was 9.55 gm. (67.2%); RBC, 3,600,000; WBC, 9,200; differential: segmented cells, 44%; lymphocytes, 50%; monocytes, 2%, and eosinophils, 4%. The red cells displayed slight anisocytosis. The urine was negative.

X-Ray.—There was cellular destruction in the right mastoid, suggestive of acute mastoiditis or, in view of the past history, of eosinophilic granuloma.

Operation.—At operation a moderate amount of pus exuded from the mastoid. Most of the cortex was eroded, and the resultant cavity was filled with pus and an elastic tumor-like tissue. The bony wall of the lateral sinus and dural plate were eroded.

Pathologic Examination.—The specimen consisted of several pieces of slimy brownish-yellowish-orange mottled tissue, the largest of which measured 2×1×0.25 cm.

Microscopic Findings: Necrosis was extensive and monopolized the picture. The surviving cells occurred in two forms. Most of these cells were large polyhedral or ovoid, pale cells, with ovoid to lobulated nuclei. The cytoplasm was moderate in amount, and the cells tended to occur in sheets. Occasional cells were binucleated. The cytoplasm of a few cells was foamy and more abundant. Mitoses were very frequent. The other type of cell

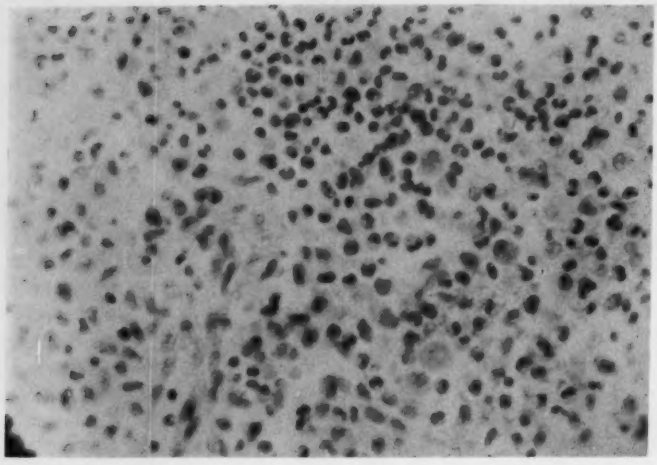

Fig. 3. — Small dark eosinophils dominate the picture. A few pale reticuloendothelial cells are present in the background.

was represented by numerous eosinophils, arranged in clusters or diffusely scattered. The picture was the same as before except that there was more necrosis and less fibrosis (Fig. 3).

Pathologic Diagnosis.—Eosinophilic granuloma of bone.

Treatment.—Six x-ray treatments were given to this area, totalling 600 r. The child was discharged as improved, on March 3, 1958, with satisfactory healing in both mastoids and without evidence of other skeletal or visceral granulomatous lesion.

The child was readmitted to the hospital, on June 2, 1958, for the removal of granulation tissue from the left external auditory canal. X-ray of both mastoids showed filling in of both bony defects. The granulations were removed, and histologic examination showed again the picture of an eosinophilic granuloma. Unfortunately, paralysis of the left facial nerve resulted, which has remained until the present time. An additonal nine x-ray treatments to the left ear and mastoid area were given, for a total dose of 900 r. He was discharged on July 2, 1958.

Since then, the child has been seen regularly in the outpatient department, with improvement of both ears and mastoids, as seen by inspection and x-ray. The facial paralysis has not improved.

Comment

History.—The concept of eosinophilic granuloma is of rather recent origin. Finzi,[5] in 1929, described a lesion of the frontal bone in a 15-year-old boy as a "myeloma with prevalence of eosinophiles," the histopathology of which corresponded to that of an eosinophilic granuloma. In 1930, Mignon[6] described an identical lesion in the left frontal bone in a child. Shairer[7] reported two cases in which he recognized a new entity, designating the lesion as an "osteomyelitis with eosinophilic reaction."

In the United States, Lichtenstein and Jaffe,[8] in 1940, presented this disease as a new and interesting entity of unknown origin, although they suggested the possibility of a virus etiology. They applied the name of "eosinophilic granuloma" to this lesion.

At about the same time, the lesion was reported by Otani and Ehrlich[9] as a distinct and separate disease, although they called it a "solitary granuloma of bone." It was soon apparent that in many cases the lesion was not necessarily solitary but was frequently multiple.

In 1942, Green and Farber[10] reported five cases of this disease and at that same time advanced the theory that eosinophilic granuloma of the bone is a benign variant of Letterer-Siwe disease and the Hand-Schüller-Christian syndrome and that the three are actually manifestations of the same fundamental dyscrasia. After some opposition this concept has found general acceptance, and, in view of this concept it is agreed that, although eosinophilic granuloma of the bone per se is generally regarded as a benign disease, in individual cases a

guarded prognosis should be given until the absence of more serious visceral lesions can be established. That there is an interesting overlapping of these three variants has been pointed out by Jaffe and Lichtenstein,[11] Green and Farber,[10] Williams, Pischnotte, Sammons, and Sokoloff,[12] and by Meyer.[13] As a matter of fact, Lichtenstein[14] has suggested that the three diseases be grouped under the designation of "histiocytosis X" and that the specific eponymic or descriptive name be added for clarification.

Etiology.—The etiology is unknown, but most workers regard the lesion as an inflammatory reaction to an unknown agent.

Incidence.—Eosinophilic granuloma of bone may occur as single or multiple lesions. Green and Farber[10] described a case with as many as 25 lesions. Eosinophilic granuloma is usually found in children and young adults and occasionally in older people. Males are said to predominate over females, in a ratio of about 5:1.

Pathology.—This is essentially a granulomatous lesion, causing bone destruction in sharply defined areas. In its early stages the lesion is usually cystic with hemorrhage and consists of soft, crumbly, yellowish-greenish-brown material owing to varying degrees of lipid infiltration and necrosis. Microscopically, one sees sheets of large pale mononuclear cells believed to be of reticuloendothelial origin, among which multitudes of eosinophils are scattered both diffusely and sparsely or in large conglomerations. Indeed, the eosinophilic infiltration may monopolize the picture to such an extent that the entire field is red. Occasionally plasma cells, polymorphonuclear cells, or lymphocytes may be found.

The reticuloendothelial cells are large and possess pale ovoid or reniform nuclei and abundant, granular, or occasionally foamy cytoplasm. These cells may or may not display cytophagocytosis.

Later these cells tend to disappear, and the lesion is believed to heal by fibrosis, before which the reticuloendothelial cells pass through a stage of so-called "lipogranulomatosis." It is also believed that healing by simple resolution is possible.

Symptoms.—The more superficial lesions produce pain, swelling, tenderness, and redness. Symptoms are usually not severe, and they may be lacking altogether. Slight fever and weight loss have been observed.

Laboratory.—Deviations from normal are minimal. There may be a moderate leukocytosis, with or without a moderate eosinophilia. Blood calcium, phosphorus, phosphatase, and cholesterol are normal. The urine is negative for Bence Jones protein.

Location of Lesion.—The lesions may be found in any bone or bones proximal to the wrists and ankles.

X-Ray.—There are well defined, ovoid to round or irregularly shaped punched-out areas of radiolucency, varying in size from about 1 to 4 cm.

Diagnosis.—Eosinophilic granuloma of the bone must be differentiated from osteomyelitis, tuberculosis of bone, syphilis, bone cyst, fibrous dysplasia, giant-cell tumor, multiple myeloma, metastatic carcinoma, osteitis fibrosa cystica, Ewing's tumor, and cholesteatoma. The x-ray picture is identical in Hand-Schüller-Christian disease, Letterer-Siwe disease, and eosinophilic granuloma of bone.

Definitive diagnosis depends, of course, upon microscopic study of biopsy material.

Prognosis.—The prognosis is excellent if one can exclude visceral involvement. However, the overlapping of eosinophilic granuloma of bone and the more serious and lethal other members of the histiocytosis X complex must be kept in mind, and, when the diagnosis of eosinophilic granuloma of bone is first made, a guarded prognosis is in order.

Treatment.—There is no specific treatment. Treatment consists of curettage and x-ray therapy, to which the lesions promptly respond. Spontaneous healing is believed possible.

Summary

A case of bilateral eosinophilic granuloma involving both mastoid processes in a 2-

year-old white boy is reported. The rarity of this distribution of the lesions is stressed. A brief review of the subject of eosinophilic granuloma of bone is presented.

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Advances in Preservation of Hearing of the Chronically Discharging Ear

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During the last century otologists have tried to eradicate infection and preserve or improve the hearing of patients affected with chronic otitis media. It appeared for some time that this goal had not been satisfactorily achieved with the frequency desired by use of the standard procedures.

The first successful attempt to eliminate infection safely and preserve or improve hearing in patients with chronic otitis media was attained through the modified radical mastoidectomy. This was first performed in the late 19th century, by Koerner [1] and Jansen.[2] Heath [3] and Bryant [4] advocated the modified radical mastoidectomy but did not define its indications. The operation as described by these authors failed to remove all the pathologic processes. However, as indicated by Moore,[25] Heath had found that inflammatory changes in the middle ear were reversible, provided patency of the Eustachian tube was respected and the tympanic cavity protected by exteriorization of the involved mastoid antrum by way of the external auditory canal.

Kopetzky [5] wrote a critique of the modified radical operation as practiced,[1-4] rejecting the procedure as having no sound surgical principles and objecting to its use in cases in which the simple mastoidectomy could accomplish the same results. Bondy [6] had the distinction of establishing the indications for operation. These are as follows: (1) good hearing, (2) pars tensa intact, (3) small perforations in Shrapnell's membrane.

Heath's operation was directed mainly to the antrum, while Bondy's modification consisted in the removal of the lateral wall of the attic, the anterior wall of the antrum, Sharpnell's membrane, and the facial bridge to the lowest limits of safety in relation to the facial nerve. In this manner all of the diseased tissue was removed and the hearing preserved. Siebenmann's [3S] modification of the radical operation consisted mainly in the Y-type of incision for the meatal flap and enough removal of the bone of the mastoid and attic area to make these parts accessible through the external auditory canal. The modified radical mastoidectomy, like all temporal bone surgery, was greatly facilitated by Lempert's endaural approach and the use of magnification.[8]

If the epitympanic pathologic process had been completely controlled when a modified radical mastoidectomy was performed, a tympanomeatal flap was not developed as recommended by House,[9] and a mastoid cavity was not created, avoiding its lifetime care and complications (Fig. 1).

In cases of extensive involvement, which are commoner, different types of rotation flaps were advocated by Baron, Kettel, and Juers [10-12] to line the mastoid cavity and seal the perforation of the typmanic membrane (Figs. 2 and 3). There are three schools of thought in connection with the management of the cholesteatoma matrix. The first advocates removal [13-17]; the second, preservation,[10,18-23] while the third (to which we belong) either removes or retains the matrix, depending upon the findings at the time of the operation.[24-26]

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The author wrote this paper while Associate Professor in the Department of Otolaryngology and Maxillofacial Surgery at the State University of Iowa.

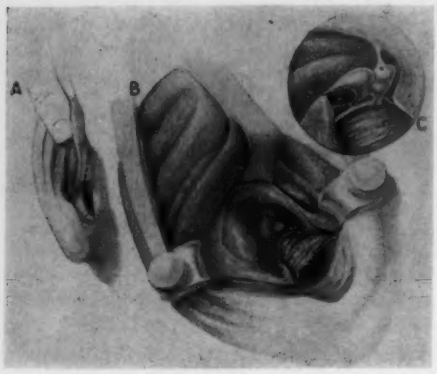


Figure 1

Fig. 1.—*A*, endaural incision. *B*, the complete removal of the mastoid cells and the exposure of the epitympanum with the visualization of the incus and head of the malleus. The thinned arch of the bridge has been partially removed. *C*, removal of the bony bridge and eradication of epitympanic pathologic process. Note that a tympanomeatal flap is not developed in this technique. Redrawn after House, H. P.: Surgery of the Chronically Discharging Ear, *Arch. Otolaryng.* 49:135-150, 1949.

Figure 2

Fig. 2.—*A*, incision made in the tympanomeatal flap and excision of a triangular section of skin in uppermost portion of flap to allow rotation of the posterior half of the flap. *B*, after the freshening of the edges of the attic perforation the posterior part of the flap is advanced downward and forward, closing the perforation. Redrawn after Baron, S. H.: Conservation of Hearing in the Modified Radical Mastoidectomy, *Laryngoscope* 64: 365-402, 1954.

In relation to the management of the incus and head of the malleus, Lathrop [16] advocated their routine removal in the modified radical mastoidectomy, while Baron,[26] following Lempert's technique, preserved the ossicles.

Moore [25] and Davison [27] stated that when the head of the malleus and incus are diseased they should be removed.

When there was disruption of the incudostapedial joint, Juers [12] advised the removal of the head of the malleus and incus while Wright et al.[28] left the ossicles in place to preserve a good air space in the middle ear, placing a skin graft against the partially destroyed long process of the incus and head of the stapes.

The histopathological processes affecting the middle ear ossicles have been reported by Grippaudo,[29] and we have been able to corroborate his observations in some of our cases.

In spite of the frequent and severe microscopic changes found in the middle ear ossicles of patients affected with chronic otitis media, we believe that these structures should be preserved when they are in continuity, since clinically some of these changes have been known to be reversible. Until we gain further knowledge on this subject and can correlate precisely our surgical and microscopic findings we should respect the continuity of the ossicular chain. But whenever there is disruption of the ossicular chain, in our opinion, it is safer and advantageous for the patient to have a carious incus removed and the bridge taken down than to perform surgical acrobatics by bringing a graft against a partially

Fig. 3.—*A*, large cholesteatomatous sac arising from an epitympanic perforation. The connection between the sac and the perforation is maintained by a stalk, seen in the diagram. *B*, incision through the tympanomeatal flap, superior aspect of the perforation, and sac of the cholesteatoma. A perpendicular incision in the flaps permits their rotation into the mastoid bowl. *C*, the cholesteatoma matrix is left in place; the perforation has been eliminated, and the flaps covered a great portion of the mastoid bowl. Redrawn after Baron, S. H.: Conservation of Hearing in the Modified Radical Mastoidectomy, *Laryngoscope* 64:365-402, 1954.

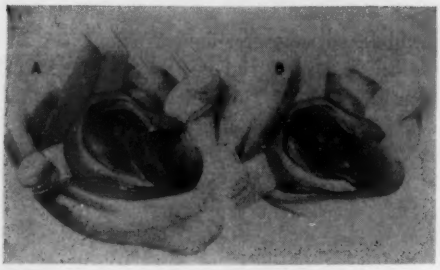


Fig. 4.—*A*, after all pathological processes of the mastoid, middle ear, incus, and head of the malleus are removed, an inferior incision is made in the tympanomeatal flap. Another incision superiorly is indicated by the dotted lines, which also includes the attic perforation. *B*, the tympanomeatal flap is slid forward and downward, closing the perforation and sealing off the tympanic cavity. The pars tensa of the drum is brought in contact with the head of the stapes. Redrawn with permission from Juers, A. L.: Preservation of Hearing in Surgery for Chronic Ear Disease, *Laryngoscope* 64:235-251, 1954.

destroyed long process of the incus and the head of the stapes. Such maneuvers violate some of the most elemental surgical principles and often lead to further surgical interventions, as demonstrated by Jongkees.[30]

If there is disruption of the ossicular chain, the incus and head of the malleus are removed. The tympanomeatal flap is tailored to close the perforation and line the mastoid bowl (Fig. 4*A*). The drum is brought in contact with the head of the stapes, as recommended by several authors [31-34] (Fig. 4*B*). The expected minimal postoperative hearing loss in these cases was 20 db. below the bone-conduction curve, according to Juers.[33] In our opinion, this is the operation of choice in cases with small- to medium-size tympanic perforations and is superior to Types II and III tympanoplasties, since the chances of a "take" are far better in a flap than in a graft. We resort to the use of a graft only when the perforations of the tympanic membrane are of such a size that they cannot be closed by sliding flaps.

The modified radical mastoidectomy was popularized by Bondy, Ballenger, Lillie, Shambaugh, and others [3,4,6,13,16,33,35-39] and achieved a greater scope by the contributions of Lempert, House, Baron, and Juers.[7-10,12] Unfortunately, the operation is best suited for patients with perforations in Shrapnell's membrane and secondary cholesteatosis or relatively small marginal perforations in the posteriosuperior quadrant of the pars tensa of the tympanic membrane.

In patients where the cholesteatoma involved the middle ear to the extent that a middle ear space could not be preserved, a radical mastoidectomy was indicated.[12]

Occasionally we were guilty of leaving granulation tissue in the ossicular chain or middle ear, with the hope of preserving the hearing. A dry ear was expected on the assumption that the major source of infection (attic and mastoid cells) had been eradicated.[10] Much to our and other's [40] chagrin, some of these patients with early successful results returned later, with a discharging ear and decreased hearing, leaving the patient with one other alternative—a radical mastoidectomy.[10]

We were unable to close 15% of large central or so-called "nondangerous" perforations of the tympanic membrane. Use of several of the nonsurgical procedures recommended,[37-45,47] or of the advocated surgical procedures [49-51] was not entirely satisfactory.

The stumbling block in the restoration of hearing and eradication of infection in chronic otitis media was greatly reduced by the work of Moritz, Thullen, Wullstein, and Zöllner.[52] They found the explanation to the well-known observation that patients sometimes developed marked hearing improvement after a radical mastoidectomy. Their explanation in these cases was that during the process of epithelization there was an accidental production of a middle ear space cavity in communication with the Eustachian tube.[53] The middle ear space was protecting the round window, the latter being separated from the oval window in such a fashion that sound waves, instead of striking both windows simultaneously, would strike them differentially. This produces motion of the inner ear fluids and vibration of the basilar membrane, accord-

ing to Bekesy's experimental work.[54] This constituted the working hypothesis of Wullstein and Zöllner.

The plastic repair of the perforated eardrum by surgical means is nothing new. In November, 1879, Berthold[55] reported two successful cases performed by him. A great deal can be learned by reviewing his paper. Conservative therapy was used until the ear was rendered dry. The deepithelization of the perforated eardrum was accomplished by the use of "English Plaster." A full-thickness graft was obtained from the inner aspect of the arm. The graft was applied to the previously prepared recipient area. These details in technique exemplify his insight in this problem. He named the operation myringoplasty.

Eli,[56] in 1880, reported nine cases in which he used a split-thickness graft, with unsatisfactory results.

Tangeman,[57] in 1883, reported his experience with this procedure, arriving at the conclusion that large grafts were not well tolerated in the ear.

The procedure fell into disrepute and did not receive proper attention until several authors reported their experiences in the current literature.[49,50] This was another step in the right direction for the preservation of hearing and the prevention of recurrent ear infections in patients with central perforations which were not amenable to conservative treatment.

Wullstein included the myringoplasty, modified radical mastoidectomy, myringostapediopexy, and fenestration operation with some basic personal modifications in a group of surgical procedures, to which he applied the term tympanoplasty.[58] Wullstein's classification is, in our opinion, excellent, because it brings to the attention of the otologist the anatomical features and physiological properties of the middle ear. His classification includes, in an orderly manner, nearly all of the surgical possibilities directed toward the preservation of hearing and eradication of infection in patients suffering from chronic otitis media. It also avoids long terms difficult to remember, such as myringostapediopexy[12] or tympanomalleolar stapediopexy[59] or myringoincudopexy.[60] These describe only some of the multiple technical details employed in the operation. Furthermore, if universally accepted, the classification will provide uniformity in nomenclature and easy comparison of reported postoperative results.

The tympanoplasty is indicated in every case of chronic suppurative otitis media, active or inactive. The only contraindications, according to Wullstein,[58] are as follows:

(1) Inner ear hearing loss to a degree that the improvement of the sound-conduction mechanism of the middle ear will be without desirable result.

(2) Complications of chronic suppurative otitis media that endanger life, such as meningitis, lateral sinus thrombosis, or cholesteatoma of the inner ear.

General criteria for operability were as follows:

(1) Adequate cochlear reserve—this being judged by pure-tone audiometry, in which air and bone measurements are recorded; speech-reception threshold; discrimination score; covering prosthesis, and/or round window-blocking prosthesis, depending upon the type of perforation[61] and acoustic measurements obtained with the acoustic probe.[58]

The experimental work of Wever, Lawrence, and Smith[62] showed that the interruption of the ossicular chain at the incudostapedial articulation, with preservation of the tympanic membrane malleus and incus, resulted in a 60 db. loss for tones of the middle range. When the eardrum and the two outer ossicles were removed, the hearing loss decreased to 45 db. The hearing loss was further reduced to 28 db. when all the structures of the middle ear, except the footplate of the stapes, were removed. From these experiments it was demonstrated that the transformer action of the middle ear accounts for 25 to 30 db. and that interruption of the ossicular chain can produce hearing loss up to 60 db. These authors also found that when the middle ear mechanism is absent the cochlea is stimulated almost as readily by the way of the round window as by the way of the oval

window. In our clinic we have been able to obtain similar measurements in patients whose incus was dislocated and in whom a tympanoplasty was later performed.

A recent report by Rosen [63] casts some doubt on the validity of the experimental measurements obtained by Wever et al.[62] and the clinical corroboration of this data. Until we gain further knowledge of the sound-conduction mechanism of the middle ear we have to accept with some reservations the values of Wever backed by similar findings.[12-64]

In spite of all the available hearing tests, the information gained is not always reliable. Such is the case when both windows are closed by middle ear pathology. In this instance, the information provided by these tests will lead one to believe that there is a cochlear involvement, whereas, in reality, the hearing loss is caused only by middle ear involvement.

(2) Patency of the Eustachian tube—politzerization and inflation of the middle ear by catherization of the Eustachian tube and x-rays of the Eustachian tube with use of radiopaque contrast media [65,66] should be a routine practice in candidates for tympanoplasties. Unfortunately, these tests, used to determine the adequacy of the Eustachian tube, are not always reliable. Negative results do not necessarily mean a tubal obstruction, since extensive pathologic process in the middle ear may prevent the entrance of air or radiopaque media into the tube or middle ear in spite of patent tubes.

Preoperative prognosis: Prediction of results in a particular case is based upon the cochlear reserve, adequacy of the Eustachian tube x-ray findings, and clinical examination of the ear under the operating microscope. The easiest cases to evaluate are those with large perforations and minicult are those with narrow ear canals, small perforations, and extensive middle ear inmal middle ear pathology, and the most diffivolvement.[61]

Types of Tympanoplasties

TYPE I (Myringoplasty).—*Specific Indications*

The specific indications are (1) central perforations of the tympanic membrane and (2) failure of closing a central perforation by conservative measures.

Prerequisites

The prerequisites are (1) adequate cochlear reserve; (2) patent Eustachian tube; (3) positive prosthesis test; (4) air-bone gap not greater than 30 db.; (5) absence of granulations, cholesteatoma, or ossicular involvement; (6) healthy skin of the external auditory canal; (7) middle ear dry from five to six months, and (8) control of nasopharyngeal and paranasal sinus pathologic processes.

As in all types of tympanoplasties, adequate conservative therapy should be used in the presence of active infection for a period of four to six weeks. Cultures and sensitivity studies from the aural discharge should be made. Administration of local and systemic antibiotics, combined with careful cleansing of the ear, will pave the way to an easier and a successful operation. Control of nasopharyngeal and paranasal sinus pathologic processes is a prerequisite to any type of tympanoplasty.

Procedure

Anesthesia: General anesthesia is preferred in all types of tympanoplasty, since the preoperative evaluation is not infallible. A more extensive operative procedure may be indicated after thorough surgical exploration of the middle ear.

Technique: Three to four centimeters of a solution of 1% procaine hydrochloride (Novocain) and 1:100,000 of epinephrine is slowly injected over the proposed site of the incision. Our incision is similar to the one advocated by Wright.[67]

Incision No. 1 is made with a Kos knife through an ear speculum. The incision is placed about 1.0 cm. posteriorly to the eardrum, beginning at 12 o'clock and ending at 6 o'clock. From the lower extremity of Incision No. 1, Incision No. 2 begins, extending through the entire length of the auditory canal. From the upper extremity of Incision No. 1, Incision No. 3 starts, extending through the entire length of the canal and the suprameatal triangle and slanting outward parallel to the helix (Fig. 5*A*). Here the Lempert spear-shaped knife is used. The incisions are made from the deep to the superficial portion because if made in the opposite direction bleeding will obscure the operative field. A conchameatal flap is developed with a sharp Kos elevator. The cartilaginous ear canal is transected with Lempert spear-shaped knife. The periosteum of the mastoid cortex is elevated with a Dean

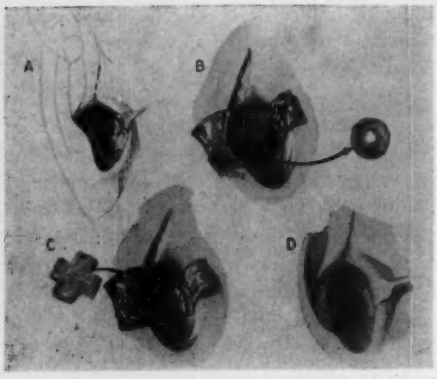


Fig. 5 (Tympanoplasty Type I).—*A*, endaural incisions used in Type I tympanoplasty. *B*, the conchameatal and tragal meatal flaps have been raised. The insert shows the removed cylinder of skin adjacent to the eardrum together with the epithelial layer of the drum. *C*, the graft is shaped to resemble the symbol of the cross and is carefully applied to the recipient area. *D*, the flaps previously developed are replaced.

periostal elevator. The conchameatal flap is swung underneath the mobilized auricle. A self-retaining retractor is placed in position. A skin flap, based laterally, is made in the anterior canal wall, as shown in Figure 5*B*.

The head lamp and × 2 magnification loupes used in the preceding part of the operation are exchanged for the operating microscope, which is now placed in position, with use of × 16 magnification. The Zöllner instruments for tympanoplasty are especially designed for work under the operating microscope, and we found them extremely helpful in this part of the operation.

The remaining cylinder of skin adjacent to the eardrum, together with the epithelial layer of the tympanic membrane, is removed in continuity (Fig. 5*B*). This maneuver is easily accomplished with the Zöllner knives. The knife should be kept superficial to the annular ligament when the epithelial layer of the eardrum is removed. The deepithelization of the tympanic membrane is carried out from the periphery to the center, to avoid enlarging the perforation.[66] As much bone as necessary is removed from the anterior and posterior canal walls. After adequate removal of the bony canal wall one is often surprised to find a larger remnant of the tympanic membrane than was suspected preoperatively.

The posterior part of the tympanic membrane is lifted out of the annulus tympanicus, as in a stapes mobilization operation. One author[66] has objected to this maneuver because, in his opinion, the tympanic membrane will lose its tension and stiffness, with the consequent reduction of its sound-pressure transformation and sound-protection characteristics. This has not been confirmed in our experience.

The ossicular chain is now examined; the round window is inspected, and, if a previously unsuspected pathologic process is found, the general plan of the surgical attack is changed accordingly. A graft 24/1,000 in. in thickness is obtained with a dermatome from the inner aspect of the arm. The accuracy in the thickness of the graft is essential to the success of the operation. We prefer a graft of 24/1,000 in. because its chances of "taking" are greater than those of the full-thickness graft.[66] With a sharp knife the graft is shaped to resemble the symbol of the cross[69] (Fig. 5*C*).

If the ossicular chain is found intact and no pathologic process is found in the middle ear, the drum is placed in its original position. In cases with large perforations, small pieces of absorbable gelatin sponge (Gelfoam) impregnated in Cortisporin (polymixin B sulfate, bacitracin, neomycin, and hydrocortisone), are placed in the middle ear to prevent the graft from sinking into it. In smaller perforations this maneuver is not necessary. In our experience and that of others,[66,69] the use of absorbable gelatin sponge in the middle ear has not been followed by serious complications.

The graft is now carefully applied to the recipient bed formed by the denuded tympanic membrane and the innermost part of the external auditory canal. It is an axiom in plastic surgery that a graft, in order to "take," requires a well-prepared recipient area and intimate contact between the graft and its bed. Omission of these details, we believe, is the commonest source of failure in this type of operation. After the graft is properly placed a sterile cigarette paper is used to cover it, as advocated by Wright,[67] and packing, similar to that used in the middle ear, is applied over the graft. The flaps previously developed are replaced (Fig. 5*D*), and Incision No. 1 is closed with fine nonabsorbable (silk) surgical sutures. Small pledgets of "Paressine lace-mesh dressing" are placed on the external auditory meatus. An external dressing is applied and left undisturbed for the next 10 days. Systemic antibiotics are also given for this length of time. On the 10th postoperative day, with sterile precautions, the pledgets and sutures are removed. The remnants of absorbable gelatin sponge, placed in the external auditory canal that have not been absorbed are removed between the 15th and 18th postoperative days.

Type II.—*Specific Indications*

The specific indications are lesions affecting the tympanic membrane, especially the pars flaccida; the tympanic cavity, especially the attic, and minor lesions of the ossicular chain.

Prerequisites

The prerequisites are (1) adequate cochlear reserve, (2) patent Eustachian tubes, (3) air-bone gap not greater than 30 db., and (4) adequate preoperative treatment to control active infection of the middle ear.

Procedure

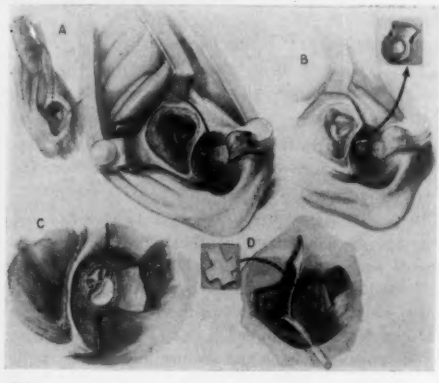
This is the same as in Type I. The incision previously described is preferred in the first two types of tympanoplasties because the bony canal wall is preserved and the long conchameatal flap serves to cover the denuded posterior osseus canal wall.

In this type of operation, because of the location of the pathologic process, the mastoid antrum is always explored, with use of House's technique,[9] and the mastoid cells are exenterated (Fig. 6*A*).

The self-retaining retractor is replaced in such a fashion that a better exposure of the ear canal is obtained. An anterior meatal flap is developed, as described in Type I. The microscope is now placed in position. The remaining cylinder of skin, epithelial layer of the eardrum, and a strip of skin corresponding to the osseous superior canal wall, are removed (Fig. 6*B*).

The epithelium of the eardrum should be removed before this structure is lifted out of the annulus tympanicus in order to take advantage of the tension of the tympanic membrane, which facilitates the deepithelization of this structure.

Fig. 6 (Tympanoplasty Type II).—*A*, insert illustrates the endaural incision used in this type of tympanoplasty. The antrum and the rest of the mastoid cells have been exenterated. The meatal flaps have been developed. *B*, insert shows the removed skin from the auditory canal together with the epithelial layer of the eardrum. *C*, the eardrum is reflected forward. The pathologic processes located in the attic are removed. The intactness of the ossicular chain is ascertained. Note that in this case the posterior canal wall is left in place. *D*, skin graft placed in position; small polyethylene tube is used to drain the mastoid cavity.



Shrapnell's membrane is also removed with impunity, since it has no importance in the sound-conduction mechanism of the middle ear.

The lateral wall of the epitympanum is removed with a burr; the epitympanum is inspected, and the presenting pathological process is carefully removed (Fig. 6*C*).

The eardrum is lifted out posteriorly from the annulus tympanicus. This maneuver is greatly facilitated if one gains access into the middle ear above the emergence of the chorda tympani nerve and continues the elevation of the annular ligament from this point downward, as recommended by Kos.[10] The intactness of the ossicular chain is confirmed; the round window and middle ear are explored, and the existing pathological process is carefully removed. Injury to the mucosal lining of the middle ear ossicles should be avoided to prevent the formation of adhesions which might interfere with the hearing at a later date.

The eardrum is replaced. A skin graft is obtained, as described in the preceding pages. The graft is shaped, as illustrated in Figure 6*D*.

The graft is now carefully placed in position, sealing the epitympanum; cigarette paper is used to cover and protect the graft, and small pieces of absorbable gelatin sponge impregnated in a solution of Cortisporin are used to pack the ear canal.

An 18-gauge polyethylene tube may be placed in the mastoid cavity and brought out through the lateral extremity of the lower incision, where it is secured with a transfixion nonabsorbable surgical suture. The wound is closed; the meatus is packed, and a dressing is applied as previously described.

The postoperative management is similar to the one recommended for Type I, with the exception that the polyethylene drain is removed on the third or fourth postoperative day.

Unfortunately, this operation is seldom indicated, since the complete removal of the pathologic processes located in the antrum often requires the removal of the facial ridge.

TYPES III, IV, AND V.—These three types of tympanoplasties are included in one group because of their similarities: A mastoid cavity is always created; the addition of a few surgical steps will lead from Type III to Type V.

Prerequisites

The prerequisites are as follows: Type III: pathologic processes affecting the tympanic membrane, tympanic cavity, mastoid cells, and ossicular chain but having a mobile stapes. Type IV: as in Type III, but with an absent or destroyed stapes. Type

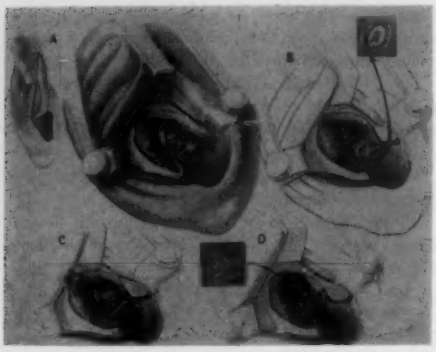


Fig. 7 (Tympanoplasty Type III).—*A*, insert: interrupted line represents the endaural incision used. The conchameatal and tympanomeatal flaps have been developed. The latter is incised in its midportion. The pathologic processes of the mastoid cells and attic are eradicated. *B*, insert shows removed cylinder of skin and epithelial layer of remnants of the eardrum removed in continuity. The tympanomeatal flaps are rotated superiorly and inferiorly. *C*, denuded tympanic membrane is reflected forward. The pathologic processes of the middle ear are eradicated. *D*, skin graft shaped and placed in position.

V: as in Type IV, together with closure of the oval window.

Procedure for Type III

With the patient under general anesthesia, a small amount of a solution of 1% procaine hydrochloride and 1:100,000 of epinephrine is infiltrated locally for hemostatic purposes. The incision used is similar to one of the several modifications of Lempert's endaural incision[10,11,71,72] (Fig. 7*A*, insert).

The prolongation of the temporal aponeurosis to the posterior membraneous canal wall is severed, as advocated by House.[9] The periosteum is elevated from the mastoid cortex with a Dean periostal elevator. A small conchameatal flap is developed, and a piece of cartilage, periosteum along with the soft tissues underlying the developed flap, is excised.

A self-retaining retractor is placed in position. The mastoid antrum is entered and exteriorized, as are the rest of the mastoid cells and epitympanum.

The skin of the posterior canal wall is elevated to a distance of about 1.0 cm. posterior to the eardrum.

With a burr, which avoids injury of the previously developed flap, as much bone as necessary is removed from the anterior and posterior canal walls.

The tympanomeatal flap is divided in its midportion, providing superior and inferior flaps, which will be used later to cover the mastoid bowl[72] (Fig. 7*A*).

The microscope is placed in position, and a circumferential peritympanic incision is made 1.0 cm. lateral to the eardrum, after which this cylinder of skin and the epithelium of the drum are removed (Fig. 7*B*).

The eardrum is lifted out posteriorly from the annulus tympanicus; the ossicular chain, round window, and middle ear are explored, and the existing lesions are corrected (Fig. 7*C*).

Note that the bridge is not taken down until we have gained information which makes its removal mandatory. This procedure is indicated when there is disruption of the ossicular chain or the pathologic process is medial to the incus. The remainder of the incus and the head of the malleus are removed. The middle ear is cleaned of granulation tissue. Utmost care should be paid to the mucosa of the middle ear. This should be traumatized to a minimum; otherwise adhesions and marked scarring will follow postoperatively.[74]

The mobility of the stapes is ascertained. The skin graft is shaped as illustrated in Figure 7*D*. The long arm of the cross will fit between the developed and displaced tympanomeatal flaps.

The middle ear cavity is filled with small pieces of absorbable gelatin sponge saturated in a solution of Cortisporin. Care should be taken not to cover the capitulum of the stapes with this packing and also to render the head of the stapes free of epithelium in cases of cholesteatoma or granulations invading this structure.

The skin graft is placed in position and protected with cigarette paper or nonabsorbable surgical sutures impregnated with Cortisporin ointment. The same packing used in the middle ear is placed over the graft.

The incision located anterior to the helix is closed with interrupted nonabsorbable surgical sutures, and two small pledgets of "Paressine lace-mesh surgical dressing" are used to keep the small conchameatal flap in position.

The postoperative care is identical to that recommended for Type I. In our study and that of others,[68,72] this seems to be the commonest type of operation.

Procedure for Type IV

This is exactly as in Type III, except that two different occurrences should be considered.

In one case, the niche of the oval window is empty but the epithelium that covers it is healthy. In this case we leave the oval window exposed and dovetail the longest arm of our graft (Fig. 8).

In other cases, the crura of the stapes are missing and the epithelium of the niche must be removed because of existing granulations.[68] In these cases the graft is placed over the niche. The mobility of the two windows in the absence of middle ear ossicles is determined by careful pressure with a blunt instrument on the sealing membrane of the

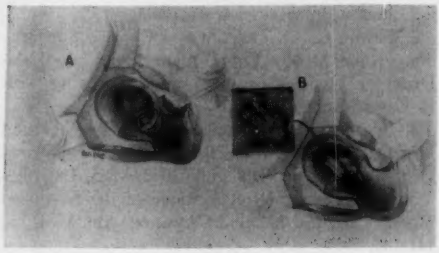


Figure 8

Fig. 8 (Tympanoplasty Type IV).—*A*, this stage is reached after steps shown in Figure 7*A*, *B*, and *C* are carried out. In this case, the epithelium covering the oval window is healthy and left undisturbed. *B*, skin graft properly shaped and placed into position.

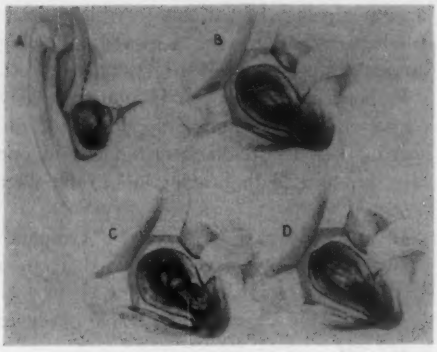


Figure 9

Fig. 9 (Tympanoplasty Type V).—*A*, endaural incision. *B*, incision of previously placed skin graft. *C*, elevation of skin graft and creation of fenestra novovalis. *D*, replacement of skin graft and completion of operation.

oval window. The pressure applied to the oval window will produce motion of the perilymph, with subsequent displacement of secondary tympanic membrane. The motion of the round window membrane can be easily observed if the niche of this window is filled with Ringer's solution.

Procedure for Type V

This is the same as in Type IV, except that three and four months after a Type IV tympanoplasty is performed, a fenestration operation is carried out (Fig. 9).

Analysis of Cases

In the past 18 months we have performed 35 tympanoplasties. In this report only the first 20 operations are analyzed, since they have been followed for more than six months from the date of operation.

Of these 20 operations, 8 belong to Type I; 2, to Type II; 9, to Type III, and only 1, to Type IV.

Because of the limited number of cases presented, this report has no statistical value, but it was thought that some information might be gained from a brief review of these cases. It is of interest to note that Type II tympanoplasty is rarely performed in our clinic. This is in accord with our policy of performing a modified radical mastoidectomy in preference to a Type II tympanoplasty whenever feasible.

The audiologic pre- and postoperative measurements for Types I and III tympanoplasties are shown in Figure 10. The

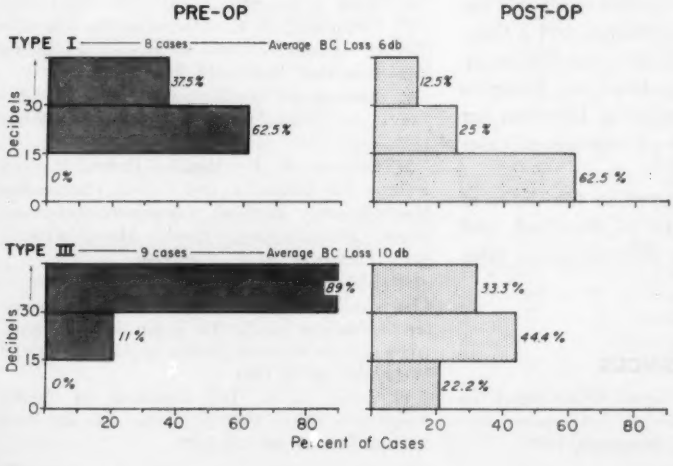


Fig. 10. — Tympanoplasty, comparison of Types I and III. The hearing measurements recorded pre- and postoperatively were obtained by calculation of the average hearing loss in decibels for air and bone conduction at 500, 1,000 and 2,000 cps.

large percentage of patients in which socially adequate hearing was achieved following surgical intervention is of special interest.

In both cases of Type II tympanoplasty we were able to bring a preoperative 45 db. hearing loss to 36 db. hearing loss in one case, and to a 27 db. hearing loss in the other case.

In our only case of Type IV tympanoplasty the hearing level improved from a 45 db. loss to a 35 db. loss.

In all of our cases we were able to obtain a dry ear.

The most frequent complication encountered in this procedure is the perforation of the skin graft. We had four such complications, giving us 20% of postoperative perforations and 80% of primary takes.

In cases where there is disruption of the ossicular chain or fixation of the stapes this complication can be avoided, according to Rambo,[76] by the use of his procedure, called musculoplasty. We do not have enough experience with Rambo's operation, and at the present time there are not enough data to judge the efficacy of this operation.

Summary

The advances and contributions of otologists in the preservation of hearing and eradication of infection in patients suffering chronic otitis media are reviewed.

A better understanding of the middle ear in sound conduction, together with the use of magnification and antibiotics and a thorough knowledge of tissue transplantation, has made it possible to direct our attention not only to the eradication of infection but also to the restoration of hearing in cases of chronic otitis media.

The technique employed in the different types of tympanoplasty is described, and the results of our first 20 consecutive cases are analyzed.

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Costen's Syndrome

A Reinterpretation

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Costen's syndrome is the term applied to a symptom-complex described by J. B. Costen in 1934.[1] The symptoms can be summarized as follows:

1. Otological symptoms: loss of hearing, stuffiness in the ears, and tinnitus aurium.
2. Head and neck pain: pain in and about the ears, headaches in the vertex and occipital regions, and pain typical of "sinus disease."
3. Miscellaneous symptoms: vertigo, tenderness of the temporomandibular joint to palpation, burning sensations in the tongue and throat, and a metallic taste.

Costen claimed [1-6] that the symptoms forming his syndrome were produced by overclosure of the mandible and that "opening the bite" would clear up these symptoms. Costen's syndrome has been debated as a single entity standing or falling by the correctness of every one of its symptoms, by the validity of the etiology given by Costen, and by the success of its treatment by "opening the bite." The literature is replete with articles pro and con, and a sampling of the more significant ones are included in the references.[1-27] I believe that one basic error has been made in all discussions of Costen's syndrome, namely, the insistence upon regarding all the symptoms as a unit and making the etiology and treatment an essential part of the acceptance or rejection of the validity of this symptom-complex.

In previous articles,[28,29] I have pointed out both the value and the importance of breaking down this syndrome into groups of allied symptoms. When this is done, a totally new picture emerges. A syndrome is a symptom-complex, a concurrence of symptoms in a disease. Etiology and treatment are not the criteria by which to judge the correctness of a syndrome. Our knowledge of the etiology and treatment of many diseases has changed over the centuries, but the description of the symptoms has often remained almost unchanged.

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History

As early as 1842, Cooper is said [30] to have described a temporomandibular joint disturbance characterized by snapping, and in 1887, Annandale opened into the joint in order to reposition the articular disk. Lang, 1909, is purported [30] to be the surgeon who introduced disk extirpation.

It was not until 1918 that any attention was paid to the role of the dental apparatus in temporomandibular joint disturbances. In that year both Prentiss [31] and Summa [32] pointed out the fact that malocclusion and loss of teeth without replacement caused damage to the temporomandibular joint. Monson,[33,34] in 1920 and again in 1921, claimed that overclosure of the mandible and malocclusion of the teeth can force the condyle back in the articular fossa, thus reducing the diameter of the auditory canal and producing deafness. Wright,[35] also in 1920, reported deafness due to malposition of the mandible.

The next significant work in this connection was done by Goodfriend, who, in 1932,[7] two years before Costen's famous report,[1] began his series of studies [7-11] which paralleled and supported Costen's theories. Costen, in 1934,[1] made his initial report on the syndrome which bears his name, and he continued writing about it with some modifications in emphasis for many years thereafter.[1-6]

Otological Symptoms

Costen [1] described loss of hearing, stuffiness in the ears, and tinnitus aurium as symptoms in his syndrome. He attributed them to overclosure of the mandible with a resultant posterior displacement of the head of the condyle. A detailed description of the anatomic explanation given by Costen and Goodfriend can be found in their papers.[1-11] The validity of Costen's anatomic explanations has been denied by many investigators,[14,20-27] but recently Young [36] has shown that lymph channels run from the internal ear through the petrotympanic fissure, the tissues of the articular fossa, and down the neck of the condyle, and Seldin [37] points out that interference with the flow of lymph by a displaced condyle or surrounding edema could produce ear dysfunction. This lymph flow could also be interfered with by spasms of the muscles surrounding the temporomandibular joint.

Loss of hearing due to mandibular overclosure certainly has not been proven. The cases reported in which there was improvement in hearing following correction of the mandibular overclosure never seem to have been confirmed by objective audiometric examinations. Hill [12] believes that the success claimed, based as it is on the subjective reports of these patients, is due to the desire of deaf people to hear better. This intense desire will cause them to report improvement for a while under any treatment. It should also be pointed out that the bias of the investigator will communicate itself to the patient and markedly influence the results. Junemann [20] found that audiometric examination failed to show any improvement in the hearing of patients in whom the mandibular overclosure was corrected. Batson [13] has never seen a clear-cut case of improvement, and Schwartz [38] goes so far as to say that he has never found the syndrome described by Costen. Costen [39] has himself admitted that "opening the bite" does not produce a lasting relief in many patients.

Stuffiness of the ears and tinnitus aurium are purely subjective symptoms which can not be measured scientifically. Since there is no objective proof of the presence of these symptoms, there can be no way to prove that the conditions have been relieved. There are no organic changes connected with these complaints, and so there must always be the possibility that they are psychophysiologic or even psychogenic.

Recently, however, I have been amazed to obtain results which would seem to cast an entirely new light on this whole problem of loss of hearing, stuffiness in the ears, and tinnitus aurium. The thinking of the investigators in this field has always been conditioned by their attitude that if overclosure of the mandible is not the cause there is no Costen's syndrome and if "opening the bite" does not clear up the difficulty conclusive proof has been obtained that there is no Costen's syndrome.

I have seen several patients in whom mild loss of hearing and stuffiness and ringing in the ears have been eliminated as a result of blocking myofascial trigger mechanisms in the mandibular musculature. These were facts mentioned by the patient without questioning and so unpremeditated that it was impossible to check the original loss of hearing with audiometric tests. This would seem to eliminate the possibility of unconsciously influencing the patient. However, any balanced assessment of these findings must certainly await more observations and study. At this time, no explanation is possible beyond the thought that the trigger area may have produced muscle spasms in a zone of reference which constricted the lumen of the Eustachian tube. This is of course a most provocative thought, and a very interesting field for investigation is offered.

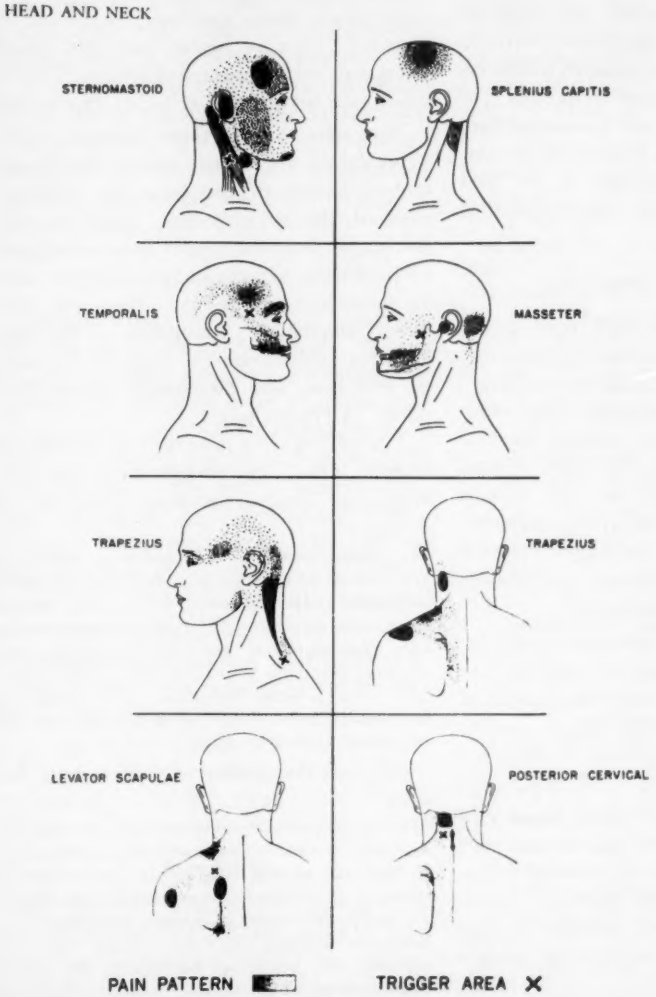
Head and Neck Pain

The head and neck pain that Costen ascribed to overclosure of the mandible is varied. Again I should like to point out that there has been a "take it or leave it" at-

titude about Costen's syndrome—either everything is correct (symptomatology, etiology, and treatment), as Costen believed, or everything is wrong and must be discarded *in toto*. This of course is ridiculous, for a syndrome is only a symptom-complex; etiology and treatment are separate and distinct from a description of symptoms.

If we view the head and neck pain of this syndrome from a point of view different from Costen's overclosure of the mandible, a new and scientifically acceptable picture emerges. While overclosure of the mandible can produce some of these pain symptoms, most of them have been shown to be unrelated. Let us examine these symptoms by themselves.

Pain in and about the ears in these patients is referred pain from either a temporomandibular joint disturbance or a myofascial trigger area. It is not the purpose of this paper to go into a discussion of these problems and their differential diagnosis, which I have done elsewhere [28,29,40-44] in detail. Travell and Rinzler [45] have found that the pain reference patterns of the



Pain-reference patterns of myofascial trigger areas in the most commonly involved skeletal muscles of the head and neck. Black areas represent the "essential" zones and stippled areas the "spill over" zones. Reproduced with permission from Travell, J., and Rinzler, S. H.: *Postgrad. Med.* 11:425, 1952; Minneapolis, Interstate Postgraduate Medical Association.

skeletal muscles can be accurately charted. In the Figure they have shown the pain reference patterns of myofascial trigger areas of the skeletal muscles of the head and neck. The pain patterns consist of two areas: the "essential" zones and the "spill-over" zones. The essential zones (which show as solid black) are those areas in which there was referred pain in 100% of the subjects. The spill-over zones (which show as stippled) are those areas in which there was referred pain in less than 100% of subjects—the more frequent the occurrence, the heavier the stippling.

Headaches in the vertex and occipital region are mentioned in this syndrome. Headaches are a complex problem which has been approached from many viewpoints.[40-42, 46-50] However, we are only concerned here with the pain involved in Costen's syndrome, and this is clearly explainable on the basis of the myofascial trigger mechanism.[40-45, 50,51]

Miscellaneous Symptoms

Vertigo was included by Costen in his syndrome, but his explanation has been disproven anatomically.[14,21-30] However, Travell and others [50,51,63,64] have shown that myofascial trigger areas can produce postural vertigo and headache. I [43,44] have pointed out that trigger areas in one muscle can produce trigger areas in other muscles, thus making it possible for trigger areas in the mandibular musculature to produce vertigo under certain conditions.

Tenderness of the temporomandibular joint to palpation may be due to an arthrosis or to the joint becoming a zone of reference for a myofascial trigger area. I have discussed this in detail elsewhere,[43,44] and it is beyond the scope of this paper.

Pain in the digastric and geniohyoid muscles is referred to the tongue, and pain in the pterygoid muscles is referred to the throat.[25] Thus, spasms of these muscles or trigger areas in them will produce the pain or "burning sensations" included in Costen's syndromes.

The "metallic taste" Costen describes is so impossible to evaluate or measure in any scientific way and so subjective, with no organic basis, that we must perforce ignore it.

The Myofascial Trigger Mechanism

The myofascial trigger area is a small, circumscribed, very hypersensitive region in myofascial tissues from which arise impulses to bombard the central nervous system and produce referred pain. This area exhibits circumscribed deep hyperalgesia, fasciculation, and referred pain, but it is only a physical sign of whose existence the patient is unaware. This mechanism has been found in many varied pain syndromes,[43-45,50-64] which are beyond the scope of this paper.

The zone of reference presents pain, hyperalgesia, and muscle spasm, and the extent of its area depends upon the sensitiveness of the trigger area. High-intensity discharges from the trigger area are capable of producing autonomic concomitants, such as vasomotor changes, sweating, and pilomotor stimulation. Stimulation of the trigger area leads to muscle spasm in the zone of reference, and this muscle spasm produces its typical pain.

Travell [51] divides the causes of myofascial trigger areas into precipitating and predisposing. The precipitating causes are as follows:

(1) sudden trauma to musculoskeletal structures, (2) unusual or excessive exercise, (3) chilling of the body, (4) immobilization, (5) an acute visceral lesion, such as myocardial infarction or appendicitis, with localized reflex spasm of the skeletal musculature, (6) acute arterial closure in the extremities, as in popliteal thrombosis, (7) rupture of an intervertebral disk with nerve root pressure, and (8) acute emotional stress.

She lists the predisposing factors as follows:

(1) chronic muscular strain, produced by repetitive movement frequently performed over a long period of time, (2) general fatigue, (3) acute infectious illness, e. g., infectious mononucleosis, acute hepatitis, or an acute upper respiratory infection (post-infectious myalgias), (4) a chronic focus of infection, (5) nutritional deficiencies, (6) a progressive lesion of the nervous system, (7) nervous

tension, (8) syndromes of the menopause and male climacteric, and (9) hypometabolism with creatinuria.

The mechanism of myofascial pain is not entirely understood, but it is a pathophysiologic disorder of some sort, possibly a feed-back neuron circuit. The important fact to us as clinicians is that it produces a self-perpetuating pain cycle. The pain itself is dull and aching in character.

Myofascial trigger areas can be activated by various stimuli, which are widespread in their occurrence; motion stretching the structure, pressure, needling, intense heat or cold, and prolonged cooling or drafts. These trigger areas often become clinically latent, but they may be activated at any time.

It is often difficult to make a differential diagnosis of a temporomandibular joint disturbance or a myofascial trigger mechanism. The details of this are beyond the scope of this paper, but I have discussed them in detail in another paper.[43]

Comment

Costen's syndrome is only a syndrome and should be evaluated as such. It is a symptom-complex and its validity should not be made dependent on proof or disproof of Costen's etiology. Certainly, there has been ample proof that overclosure of the mandible will not produce the entire symptom-complex. However, the myofascial trigger mechanism can and does produce all the symptoms, even though the exact mechanism involved is not understandable with our present knowledge.

Costen's syndrome has fallen into disrepute in recent years because the etiology and treatment suggested by him have proven incorrect. I feel that this disrepute is unwarranted in the new light shed by the understanding of the myofascial trigger mechanism.

Summary

A different etiological approach to Costen's syndrome is offered, together with some new evidence to indicate its validity. It would appear possible that Costen's syndrome may again find its place in scientific medicine, although with a new etiology and new anatomical explanations, some of which are not as yet forthcoming.

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Otogenic Meningitis in Infancy and Childhood in the Antibiotic Era

Observation on Thirty-Eight Children Treated at Los Angeles County General Hospital from 1950-1957

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A survey of current literature reveals a significant reduction in the incidence of otogenic complications.[2] This is universally attributed to the use of antibiotic and chemotherapeutic agents.[3,5,9,11,12] The purpose of this paper is to call attention to the fact that the problem of otitic meningitis in this antibiotic era has not fully been solved. This is particularly true in infants and young children. The mortality rate still remains high in this age group. Successful conduct of suppurative meningitis requires a concerted effort of both the public and the practitioners of medicine. The public must be conditioned to seek medical aid promptly. The key to an early diagnosis is in the hands of the general practitioners and pediatricians. They are likely to see the child with meningeal symptoms first. They must be ever meningitis conscious. However, the otologist must share his responsibility in order to reduce this high incidence of morbidity and mortality in this serious illness.

During an eight-year period, from 1950 to 1957, thirty-eight cases of purulent otitic meningitis in infants and children under 13 years of age were observed. These were either secondary to acute suppurative otitis media or concurrent intracranial infection with acute otitis media. An attempt is made in this paper to review salient points of interest in these 38 children. Patients with tuberculous meningitis were not included.

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TABLE 1.—*Age Distribution and Mortality Rate*

	Age, Yr.				
	Under 1	1-2	2-5	5-10	10-13
Total cases	19	4	7	5	3
Deaths	12	2	3	2	0
Recovered	7	2	4	3	3
Mortality	63%	50%	43%	40%	0

Cases of chronic meningitis and various forms of its complications were also excluded.

Table 1 indicates the age distribution and mortality rates in various age groups. The highest incidence of otitic meningitis occurred under one year of age. The mortality rate was 63%. The high incidence of mortality in this age group is attributed to a combination of several factors. Among them are (1) the anatomical peculiarity of the middle ear cleft, which permits easy access of infection to the intracranial areas*; (2) the lack of natural immunity; (3) the difficulty in accurately evaluating the otoscopic changes in the middle ear, and (4) the altered behavior of bacteria when inadequate and inappropriate drugs are given.

Sex and Racial Distributions

Sex and racial distributions of these 38 children are shown in Table 2. Mexicans

* Jakabfi[8] enumerates several pathways of infection. These are: (1) persistent fissures; (2) dehiscences, particularly above the tegmen; (3) the labyrinth, with its various communicating ducts and canals; (4) hiatus arcuatus; (5) the apex of the pyramid, along the facial nerve and the communicating vessels which are richly endowed in infancy and early childhood.

TABLE 2.—*Sex and Racial Distribution*

	Race									
	All		Caucasian		Negro		Oriental		Indian	
	M	F	M	F	M	F	M	F	M	F
Total cases	24	14	21	14	1	0	1	0	1	0
Deaths	12	6	9	6	1	0	1	0	1	0
Recovered	12	8	12	8	0	0	0	0	0	0
Mortality	50%	43%	43%	43%	100%	0	100%	0	100%	0

are included among the Caucasians. The series corresponds to the general distribution of the racial groups in this area. Meningitis spares no race.

Clinical History on Admission

The duration of meningitis prior to hospital admission is difficult to assess. Among those recovered, 4 were recorded as having been sick 24 hours before entry, 10 were reported to have been sick 2 to 4 days, and 5 were stated to have been sick longer than 5 days prior to admission. Among those who died, two were recorded as having been well 24 hours before admission, eight were sick 2 to 4 days, and nine were sick 5 days or longer before being admitted to the meningitis ward. Duration of meningitis prior to admission is shown in Table 3. It will be seen that the earlier the institution of treatment the better the final outcome, the rate of recovery being highest in the 24-hour group and worst when treatment was instituted five days or longer after the initial symptoms developed.

Gastroenteritis and Otoantritis

Among those 19 who died, there were 3 children who had developed diarrhea, vomiting, and dehydration prior to admission. The presence of acute otitis media was totally overlooked in these three before their entry. They died within 24 hours, in the hospital, from extreme toxemia and dehydration. Whether the gastrointestinal disorder preceded otoantritis or otoantritis precipitated diarrhea, vomiting, and dehydration or whether these two conditions are independent of each other is difficult to determine in a given case after meningitis is well established. Failure to discover otitis media in its early stage must be recognized as a distinct factor in the development of intracranial complication in an infant whose vitality is greatly reduced by overwhelming toxemia.

TABLE 3.—*Duration Prior to Admission*

	Duration			
	24-Hr.	2-4 Days	5 Days or More	Total
All cases	6	18	14	38
Deaths	2	8	9	19
Recovered	4	10	5	19
Mortality	33%	44%	64%	

CASE 1.—An 8-month-old Caucasian girl developed fever, vomiting, and diarrhea five days before entry. Her family doctor gave here penicillin, and there was temporary improvement. On the day of her admission she ran a temperature of 105 F. No record was made as to the condition of her ears. She was extremely dehydrated. Upon entry the spinal fluid cell count was 11,000, with 62% monocytes. She remained comatose, and her course in the hospital was progressively downward. She died within 24 hours after entry. Necropsy indicated the meninges were free from distinct exudate, but there was venous congestion and an occasional terminal hemorrhage. The left ear contained pus, and there was acute mastoiditis. The nerve cells indicated toxic degeneration.

Comment.—This is a rare instance in which there was unilateral otoantritis. In the two others in this series bilateral purulent otoantritis was present. This is the usual finding at necropsy.

Upper Respiratory Infection

In 8 children among 19 who died in the hospital, meningitis was ushered in with the

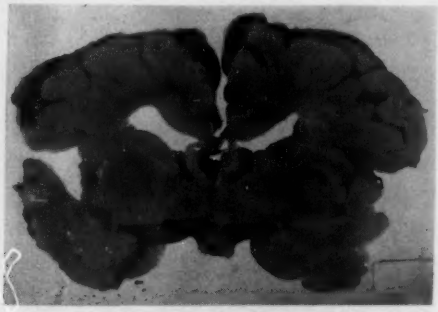

Fig. 1.—Enlargement of the lateral ventricles secondary to purulent otitic influenzal meningitis in a 3-month-old Negro girl.

symptoms of upper respiratory infection of one to six days duration before admission. Rhinorrhea and nasal obstruction masked the early signs of otitis media. They were brought into the hospital after the signs of meningitis were well established.

CASE 2.—A 3-month-old Caucasian boy caught a cold three days or longer before admission. He was a seven-month premature baby, one of twins. His abdomen became distended and was unrelieved by enemas. He refused feedings. He remained critically ill in the hospital. Both drums were dull, but myringotomy was performed very late. The spinal fluid was glassy in appearance, and Diplococcus pneumoniae (pneumococcus) was recovered. The child died within 24 hours. Necropsy showed the meninges were swollen and contained cloudy fluid. Both middle ears were filled with whitish exudate.

Comment.—Prematurity, associated with its usual inadequate natural immunity, plays a vital part in unfavorable outcome. This case was no exception. When the treatment is delayed, the child dies from overwhelming toxemia and brain injury.

Irritability, Nausea, and Vomiting

In 7 children, in this group of 19 who died, irritability, nausea, and vomiting were the prominent initial symptoms. Thus the presence of acute otitis media was completely overlooked or discovered very late in the course of the disease.

CASE 3.—A 20-month-old Mexican girl had pneumonia at the age of 6 months. The child developed a temperature of 103 F. Thirty hours before she was admitted she developed nausea, vomiting, and increasing irritability. She was stuporous on entry. No record was made as to the condition of the ears. Her spinal fluid yielded Meningococcus. There were 11,200 cells, with 95% polymorphonuclear cells. In spite of intense parenteral fluids, numerous antibiotics, and whole-blood transfusions, she died on the second day from overwhelming meningococcemia. Necropsy indicated the patient died from fulminating meningitis. There were a few scattered subarachnoid hemorrhages. Both middle ears contained stringy seropurulent exudate from which Diplococcus meningococcus was recovered.

CASE 4.—A 3½-year-old Caucasian physician's daughter developed fever four days prior to admission. On the second day of her illness she was seen by another physician. A diagnosis of acute tonsillitis was made, and she was placed on antibiotics. The following day her temperature was normal. On the morning of her admission she awoke with a headache, nausea, and stiffness of her neck. Again she was seen by the physician, who discontinued antibiotics because of nausea and vomiting. She slept all day. Because of the development of symptoms suggestive of meningeal irritation, she was admitted to the hospital that evening with a temperature of 101.6 F. Her spinal cells numbered 1,400, with 99% polymorphonuclear cells. The organism failed to grow on culture. She responded well to chemotherapy. In two days she was afebrile. Her ears appeared to be normal. However, x-rays showed early acute right mastoiditis. On her fourth day in the hospital she had convulsions and died of respiratory failure. Necropsy indicated minimum changes in the meninges, but the brain was edematous. There was active toxic nerve degeneration.

Comment.—Even under the most favorable circumstances the symptoms may be masked and mislead one to delay the diagnosis.

Convulsions

Nine children in this series had one or more convulsive seizures either before admission or during their study in the hospital. All except one died, indicating that convulsions portend a poor final outcome.

CASE 5.—A 4-month-old Japanese boy caught cold 10 days before entry. He developed fever and right-sided convulsions two days prior to admission. His ear drums were dull and hyperemic. His pharynx was slightly injected. The spinal cell count was 1,225, and the culture yielded Hemophilus influenzae. His temperature in the hospital fluctuated; on the 10th day he developed a series of convulsions, and he died the following day. Necropsy indicated suppurative meningitis associated

Fig. 2.—Purulent otitic meningitis in a 4-month-old Japanese boy, showing heavy exudate on basilar surfaces of the frontal and temporal lobes.

Fig. 3.—Early otitic pneumococcic meningitis in an 18-month old Caucasian boy, showing meningeal congestion at base of brain.

with subarachnoid hemorrhage and acute cortical changes. Purulent exudate was found in both ears.

Head Injuries

Histories of head injuries were recorded in two children. The first child developed fatal recurrent meningitis after a second head injury. The second child recovered. Head injuries, however trivial, may develop into serious complications. X-rays may or may not reveal fracture lines. In most instances a break occurs in soft tissues, which permits the passage of the organism.

Case 6.—An 18-month-old Caucasian child had fallen on his head five months prior to entry. He developed meningitis and was successfully treated in another hospital. He fell again on his face two days before admission. Recurrent meningitis developed, and he died within 24 hours. Necropsy indicated left petrositis secondary to latent acute mastoiditis, purulent meningitis, and generalized cerebral edema. Pneumococcus was recovered in the spinal fluid and in the left middle ear.

Diagnosis

Diagnosis of meningitis is made from isolation of the causative organisms and cellular enumeration of the cerebrospinal fluid. In the present series D. pneumoniae was isolated in 11 children, and 8 of these died. H. influenzae was accounted for in five cases, and four of these died. Neisseria meningitidis (meningococcus) was isolated in six children, and two of these died. In three Staphylococcus pyogenes var. aureus was recovered from the spinal fluid, and an uneventful recovery was made. In 13 children turbid spinal fluid was recovered, but the organisms failed to grow on the usual culture media. In younger children gastrointestinal disorders and nasal obstruction overshadowed the presence of acute otitis media and the initial symptoms of meningitis. It should be remembered that persistent headache, pyrexia with or without remission, progressive nuchal rigidity, and vomiting with no relation to diet are signs of meningeal irritation. One should be constantly alert to recognize these symptoms.

Pathology

Necropsy examination was performed on all children who died in this series. Such an examination afforded an opportunity to

TABLE 4.—*Organisms*

	D. Pneumoniae	H. Influenzae	N. Mengitidis	S. Aureus	Unknown	Total
Total	11	5	6	3	5	38
Deaths	8	4	2	0	5	19
Mortality	73%	71%	33%	0	38%	50%

inspect the residual lesions in each case. In the immediate proximity of larger vessels, where the antibiotics came in contact with the exudate, a clear zone of resolution occurred. Where the exudate was too thick and the antibiotics failed to penetrate the exudate, there were always "islands" of isolated patches. In favorable cases it is assumed that these "islands" progressively reduce in size until final resolution develops and the patient finally recovers. When inadequate or inappropriate therapy is given, endotoxin and exogenous toxin are diffusely distributed and the patient succumbs to overwhelming toxemia.

Infective Organisms

The types of organisms that were encountered and the mortality rate of each are indicated in Table 4. Beveridge[1] recorded his observation of 110 infants and children with suppurative meningitis, for a three-year period. His recovery rate in 28 cases of pneumococcic meningitis was 60% and in 70 cases of influenzal meningitis was 83%. Smith[13] reviewed 409 cases of purulent meningitis in infants and children for a ten-year period. The recovery rate with pneumococcic meningitis was 85.4%; influenzal meningitis, 92.5%; meningococcic meningitis, 84.4%, and among 24 patients in whom organisms could not be isolated, 56%. Joos and Leeper,[7] of the University of Michigan, observed 38 cases of bacterial meningitis in children under two years of age in a five-year period. Thirteen of these had Hemophilus influenzal meningitis, with a mortality rate of 38%. Twelve had meningococcic meningitis, with a death rate of 33%. Three cases of pneumococcic meningitis also had a mortality rate of 33%.

Treatment

Comprehensive discussions on the treatment of suppurative meningitis have been given by McBean,[10] Etteldorf,[4] Yow,[14] and others. Responsibility of medical management of meningitis rightly belongs to pediatricians, but the otologist should be familiar with the problem. As a consultant, he should be prepared to render whatever service is required of him in his field.

Successful management of otitic meningitis depends on (1) early diagnosis; (2) institution of steps to eliminate the offending organisms in the primary focus of infection in the middle ear and the mastoid cavity, including the petrous pyramid if it is involved; (3) maintenance of optimal electrolytes; (4) supportive measures to combat the effect of both endotoxin and exogenous toxin, and (5) reduction of every chance of permanent injury to intracranial structures.

To attain the objectives above, a general plan of treatment has been formulated, with individualization of therapy to meet each patient's requirements.[8] Upon admission the patient is placed in the isolation ward. Blood cell count, cultures, spinal fluid examination, and urinalysis are performed. These are repeated during his hospitalization as often as indicated.

When otologic disorders are suspected, the patient is seen by the attending otologist for consultation. Myringotomy was performed on 16 children; of these, six died. Since the introduction of antibiotics and newer chemotherapeutic agents the behavior of the organisms in the respiratory passage has been markedly altered. The tympanic membrane no longer presents the typical textbook appearance in many instances of infection in the middle ear. Otoscopic views are often misleading. Myringotomy is a

simple surgical procedure, but it is a life-saving measure when indicated. A cortical mastoidectomy is seldom required in children.

In 1953, broad-spectrum antibiotics became available. They have been freely used in conjunction with sulfonamide, penicillin, and others. Intrathecal medication is not practiced. The optimum blood concentration of sulfonamide is 10 to 12 mg. % and spinal concentration 12 to 20 mg. %. Urine pH 7 is maintained to prevent formation of sulfonamide crystals by adequate hydration.

To combat anemia and overwhelming toxemia, whole blood and plasma were judiciously used. Cortisone in various forms was the drug of choice for treatment of shock. Barbiturates and chlorhydrates were employed for sedation. After the treatment had been discontinued and the patient remained afebrile, those with uncomplicated meningococcic meningitis remained in the hospital 5 to 7 days; those with influenzal meningitis, 10 to 14 days, and those with pneumococcic and all other meningitis, 7 to 10 days.

Summary

Thirty-eight cases of otitic meningitis in children, treated in a large tax-supported general hospital from 1950-1957, are reported.

Meningitis was secondary to acute otitis media or concurrent with acute otoantritis.

Masking of symptoms of acute otitis media was the chief cause of delay in diagnosis. This in turn contributed to a high mortality rate in this series.

The key to the solution of reduction of morbidity and mortality of otitic meningitis is earlier recognition of the meningeal symptoms and prompt institution of adequate and appropriate chemotherapy.

When hospital treatment was instituted within 24 hours after the onset of meningitis, the recovery rate was 67%. When this was delayed two to four days, the recovery rate was 56%. In the third group, in which treatment was not given until five days or longer, the rate was reduced to 36%.

436 S. Boyle Ave. (33).

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Ototoxicity of Quinine in Experimental Animals

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Quinine is a protoplasmotic poison which, in cases of cinchonism, produces tinnitus, hearing loss, dizziness, blurring of vision, photophobia, and hypotension. Administration of a large dose may produce delirium, unconsciousness, respiratory failure, and death.

Ototoxicity of quinine seems to be the result of administration of a large dose or prolonged treatment, or it may be a matter of idiosyncrasy. Hearing loss of conductive type is said to occur, by Falbe-Hansen,[1] after several clinical and experimental observations, while Forbes [2] and Rüedi et al.[3] believed it is of perceptive type.

The opinions concerning the location and nature of cochlear lesions as observed in experimental animals may be divided in two groups. The first group claims that quinine produced changes in the primary cochlear neurons and hypotonic degeneration in the cochlear receptor. The features and explanation of this phenomenon were put forward by Wittmaack,[4] and have been supported by the work of Falbe-Hansen [1] and Mygind.[5] According to this theory,[4] the hypotonic degeneration is a vital phenomenon characterized by collapse of Reissner's membrane and compression of the organ of Corti. This phenomenon is not exclusive to quinine poisoning, because it may appear in control animals fixed with intravital perfusion or postmortem fixation [1,5] and had been frequently found in human temporal bones.[6] There are some observations which suggest that hypotonic degeneration—or the so-called endolymphatic compression of Mygind [5]—is an artifact.[6,7] The second group supports the findings of Covell,[8,9] who demonstrated degenerative changes in the stria vascularis, external hair cells, and primary cochlear neurons. These findings have been substantiated by Rüedi et al.[3]

The present investigation was undertaken with two purposes. The first was to study the differential sensitivity of the cell population of the inner ear to a small dose of quinine injected in the middle ear cavity. The rationale was that, although the absolute dose of injected drug was small, the dose relative to size of inner ear was large. We assumed that a certain amount of drug will penetrate through the round and oval windows into the inner ear before it is eliminated by absorption or drainage through the Eustachian tube. We expect that this local application of the poison may produce changes more easily recognized than in lethal or protracted doses given systemically. The procedure may help in determining the location of the lesion and whether this is a degenerative change, as claimed by Covell,[8,9] or as hypotonic degeneration, as claimed by Wittmaack.[4] The second purpose was to study functional disturbances, both vestibular and cochlear. Correlation between cochlear function and histopathologic findings may help in the understanding of the type of hearing loss which quinine would most likely produce. Similarly, correlation of vestibular function and histology may help in explaining vestibular symptoms which may appear in some subjects after injection of quinine.

Method

The experiments were carried out in young, healthy guinea pigs anesthetized with ether. The operation consisted in exposing the bone of mastoid antrum and drilling a hole in it. For administration of drug, a 24-gauge hypodermic needle was fitted in the hole and the fluid injected slowly to

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avoid rupture of tympanic membrane. The operation was performed under aseptic conditions on one ear only of each animal.

Two quinine salts were used: a solution of 1% quinine hydrochloride in distilled water (14 animals) and a solution of 0.8% quinine sulfate in 95% alcohol (10 animals). In both cases, quinine hydrochloride and quinine sulfate, the amount of solution injected was 0.10-0.12 cc.; thus the quantity of the drug was about 10^{-3} gm. and 8×10^{-4} gm., respectively.

After recovery from anesthesia, behavior and reflexes, and sometimes rotatory tests, were carried out for several hours during the first day and periodically afterward. The animals were killed at convenient intervals ranging from acute preparations to 76 days.

Cochlear responses were measured in 13 animals. In the other 11 guinea pigs, cochlear function was not tested because of extensive lesions in the middle ear or death. The technique for implanting electrodes, the type of stimulus, and the procedure of recording are described elsewhere.[10]

All animals were killed by intravital perfusion with Heidenhain-Susa fluid, and both temporal bones removed in one block. The histologic technique followed is described elsewhere.[11] The serial sections were studied under ordinary light microscopy.

Results

A. Vestibular Reactions.—The animals awoke from ether anesthesia within 10

Fig. 1.—Righting reflex in a guinea pig 30 minutes after injection of quinine hydrochloride into the right middle ear The upper photograph shows the body hanging freely over the right side. The animal remained steadily in this position. The lower photographs show the righting reflex when body hangs freely over the left side.

minutes after the operation, showing signs of unilateral labyrinthectomy, that is, spontaneous nystagmus and head nystagmus to the intact side and rotation and/or rolling to the operated-on side, as well as tilting of the head. Any change in this position of the head elicited a labyinthine righting reflex, which tended to restore the tilting position. This reflex was very obvious when the animal was held by the loins with the body hanging freely. If the operated-on side was down, no reaction was produced, but if the intact side was down, the animal lifted both head and body up. These two attitudes are illustrated in Figure 1.

The progression reactions are difficult to observe in guinea pigs. Many animals showed absence of toe-spreading reflex in the hind leg corresponding to operated-on ear, while, on the opposite side, this reflex was present. Also, during lifting, the homolateral hind leg remained extended while the contralateral was flexed.

The rotatory test, performed in some animals a few hours after the operation, showed canal paralysis of the operated-on ear. Rotation toward this side enhanced the spontaneous nystagmus, while rotation to the intact side did not modify it.

The vestibular signs were essentially the same in all animals that received either quinine hydrochloride or quinine sulfate. Those treated with the sulfate compound showed less activity for some hours after the operation. This was probably due to an additional effect produced by absorption of alcohol.

The follow-up showed that all vestibular signs remained unchanged for four to five hours and thereafter subsided gradually. Within 24 hours, spontaneous nystagmus, head nystagmus, rotation, and rolling disappeared and progression reflexes were apparently normal. Many animals, which received quinine sulfate, had postural nystagmus in lateral positions, and its direction was consistently the same as the initial spontaneous nystagmus. The postural nystagmus was stronger when animal lay on the un-operated-on ear, lasted between one and two minutes, and could be reproduced at will. Its amplitude and duration subsided gradually and finally disappeared about six days after the operation.

Several animals also showed asymmetric reactions in the rotatory test for a few days, that is, rotation toward the operated-on ear gave longer and stronger reactions than rotation toward the un-operated-on side.

B. Histopathologic Studies.—The microscopic observation of 24 quinine-injected ears showed that both quinine sulfate and quinine hydrochloride produced the same type of damage. In the ears into which the sulfate compound was injected, however, the lesions were more extensive.

1. Changes in Middle Ear: Within 48 hours, the epidermis of tympanic membrane became hyperplastic. Perforations of membrana propria occurred in several animals. Chronic preparations were characterized by proliferation of fibrous and bony tissues in hypotympanum and/or attic. Both fibrous and bony tissues may involve oval and round windows and ossicular chain to the point that ossicles cannot be identified. The proliferation of fibrous tissue, in combination with infection, produced, in one case, a sequestrum of the cochlea (Fig. 2*D*).

Three control ears presented new bone formation in the hypotympanum without infection or lesions which might explain it. There is the possibility that quinine was eliminated from the quinine-injected ear through the eustachian tube and reentered the tympanic cavity of the opposite side, thereby giving rise to a bony reaction.

2. Changes in the Cochlea: Scalae. In both acute and chronic preparations, small amounts of precipitates and blood were found in perilymphatic and/or endolymphatic spaces. In one case the stria vascularis was identified as the locus of hemorrhage, while in the others the locus was uncertain. Three ears presented signs of infection associated with otitis media.

The chronic preparations showed more or less proliferation of bone and/or fibrous

Fig. 2.—Varying degrees of fibrous and bony tissue proliferation in the first turn of the cochlea, near round window. *A*, chronic experiment, 22 days after injection of quinine sulfate. Dense fibrous tissue occupies scala tympani for about 4 mm. Spots of new bone as arrow-points were also found. *B*, 76 days after injection of quinine sulfate. Partial ossification of round window membrane and fibrous tissue in scala tympani. *C*, 70 days after injection of quinine sulfate. Ossification of round window and scala tympani for about 4 mm. Fibrous tissue also found in scala vestibuli. *D*, 38 days after injection of quinine sulfate. Sequestrum of the cochlea. Dense fibrous tissue and severe infection. Hematoxylin and eosin; *A*, *B*, and *C*, × 250; *D*, × 23.

Fig. 3.—Changes in stria vascularis after injection of quinine into the middle ear. *E*, control. Hematoxylin and eosin; × 250. *F*, 1 day after injection of quinine hydrochloride. Cellular and intercellular edema and globular formation over the surface of stria vascularis. Mallory; × 250. *G*, 1 day after injection of quinine hydrochloride. Cellular and intercellular edema, globular formation on surface, and partial detachment of stria vascularis. *H*, 11 days after injection of quinine sulfate. Complete necrosis of stria vascularis. Some debris found in cochlear duct. Hematoxylin and eosin; × 250.

tissue localized to the first 4 mm. of scala tympani. These findings are illustrated in Figure 2. In some cases both fibrosis and new bone formation seemed to arise from endosteum of the cochlea, while in others fibrous and bony tissues of middle ear invaded the cochlea through round window.

Stria vascularis. The stria vascularis of all acute preparations presented cellular and intercellular edema and globular formation on the surface. Some specimens also showed vasodilation. These changes were observed in the basal turn but were found also throughout the cochlea without visible damage in other structures. In two specimens, the stria vascularis near the round window was partially detached. These acute reactions, which are illustrated by parts *F* and *G* of Figure 3, lasted for about three days. After this time, edema, vasodilation, and globular formation were not seen.

Several chronic preparations showed absence of stria vascularis adjacent to the round window (Fig. 3*H*). Otherwise, the epithelium was histologically comparable to the control side. Absence of stria vascularis seemed to be the last stage of detachment observed in acute preparations.

Spiral ligament. Some acute preparations presented pyknosis in the cells of the spiral ligament adjacent to the round window. In this region, the cells of spiral prominence may become pyknotic and may also be detached.

In several chronic preparations, the area of spiral ligament adjacent to the round window was acellular.

Limbus. The limbus, particularly, was vulnerable. The cells become firstly pyknotic, and later the nuclei disappear, leaving an acellular fiber system. The interdental groove cells may or may not be involved. The lesion most frequently was localized in the basal turn but may be found at some spots along the cochlea without being associated with changes in other structures. These findings are illustrated in Figure 4.

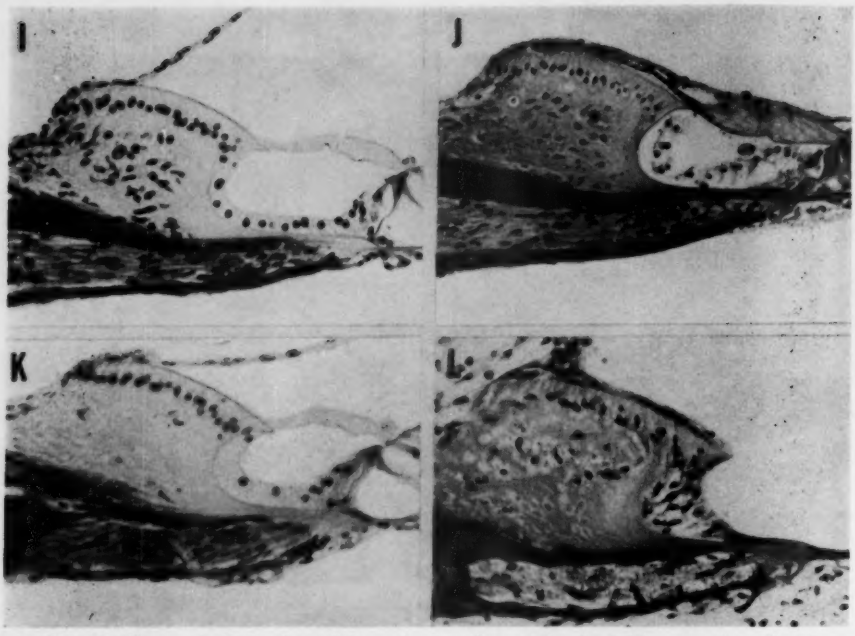
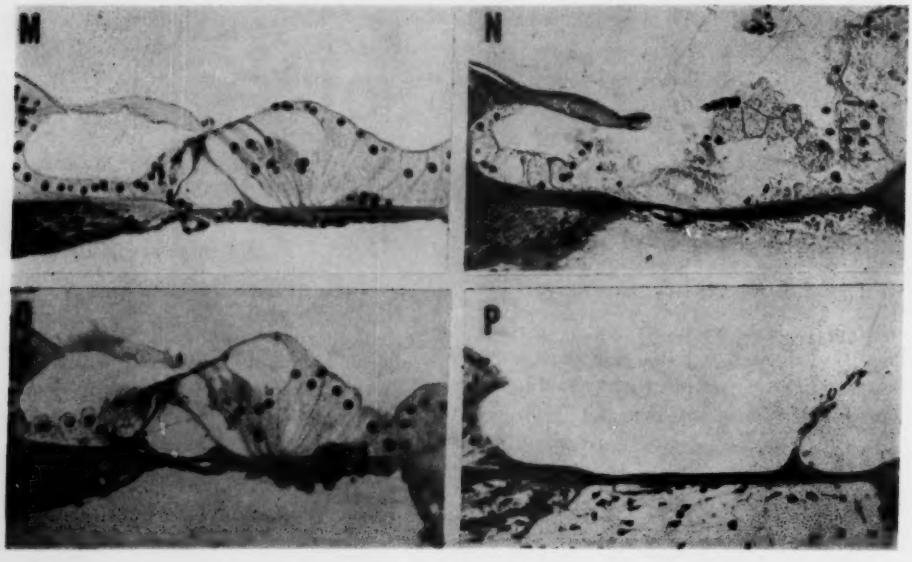


Fig. 4.—Changes in limbus by quinine poisoning. *I*, control animal. Hematoxylin and eosin; × 250. *J*, 2 days after injection of quinine sulfate. Pyknosis of all cellular elements, including interdental groove cells. Hematoxylin and eosin; × 250. *K*, 8 days after injection of quinine hydrochloride. Interdental groove cells were not affected, but nuclei in substantia propria are absent. *L*, 8 days after injection of quinine hydrochloride. Only few pyknotic nuclei remained and tectorial membrane was disintegrated, hematoxylin and eosin; × 250.

Fig. 5.—Changes in organ of Corti by quinine poisoning. *M*, control animal. Hematoxylin and eosin; × 250. *N*, 1 day after injection of quinine sulfate. Disintegration of organ of Corti. Mallory; × 250. *O*, degenerative process, indicated by absence of nuclei in external hair cells. The other cell population of organ of Corti is well preserved. Hematoxylin and eosin; × 250. *P*, 11 days after injection of quinine sulfate. Complete necrosis of organ of Corti and replacement by a flat epithelium. Hematoxylin and eosin; × 250.



Organ of Corti. In one specimen, the cells of the organ of Corti presented no histologic damage.

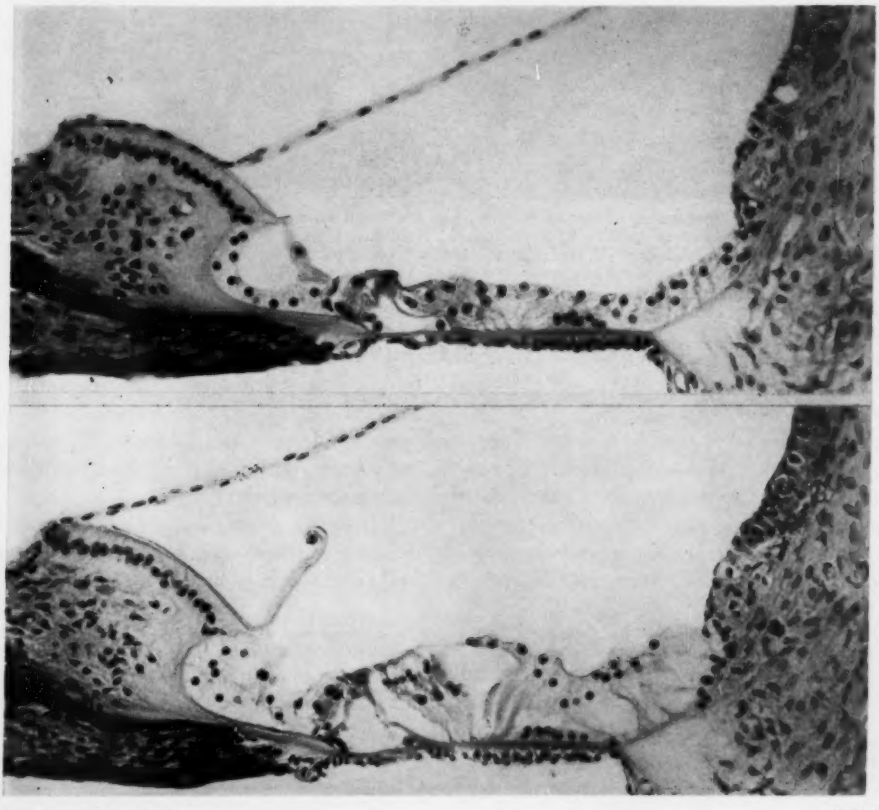
Ten specimens showed degeneration of external hair cells localized in the first 4 mm. of cochlear duct (Fig. 5*O*). Degeneration consisted in absence of nuclei, homogenization of cytoplasm, and distortion and/or absence of external hair cells. This lesion was found in acute as well as chronic preparations. The supporting cells and inner hair cells of these 10 specimens showed no detectable abnormalities.

Thirteen specimens showed detachment and disintegration of organ of Corti (Fig. 5*N*). The lesion most frequently was localized in the basal end of the first turn, but in a few cases it was extended to the second turn or throughout the cochlea.

In chronic preparations the debris was eliminated, and organ of Corti may be replaced by a flat epithelium (Fig. 5*P*). Above the necrotizing lesion the external hair cells were found degenerated for a few millimeters, while internal hair cells and supporting cells were remarkably preserved. Pyknosis or absence of mesothelial cells and disintegration of tectorial membrane were associated with necrosis of organ of Corti. Examples of disintegration, degeneration, and absence of organ of Corti are presented in Figure 5.

The 24 quinine-injected ears and 24 controls were assessed for compression of the

Fig. 6.—Compression of organ of Corti. Guinea Pig D-51, two days after injection of quinine hydrochloride in right ear. Upper photomicrograph, right ear. Reissner's membrane collapsed over limbus and tectorial membrane over a flattened organ of Corti. Lower photomicrograph, left ear. No compression of organ of Corti, but this presents advance postmortem changes which are due to failure of intravital perfusion. Hematoxylin and eosin; × 250.



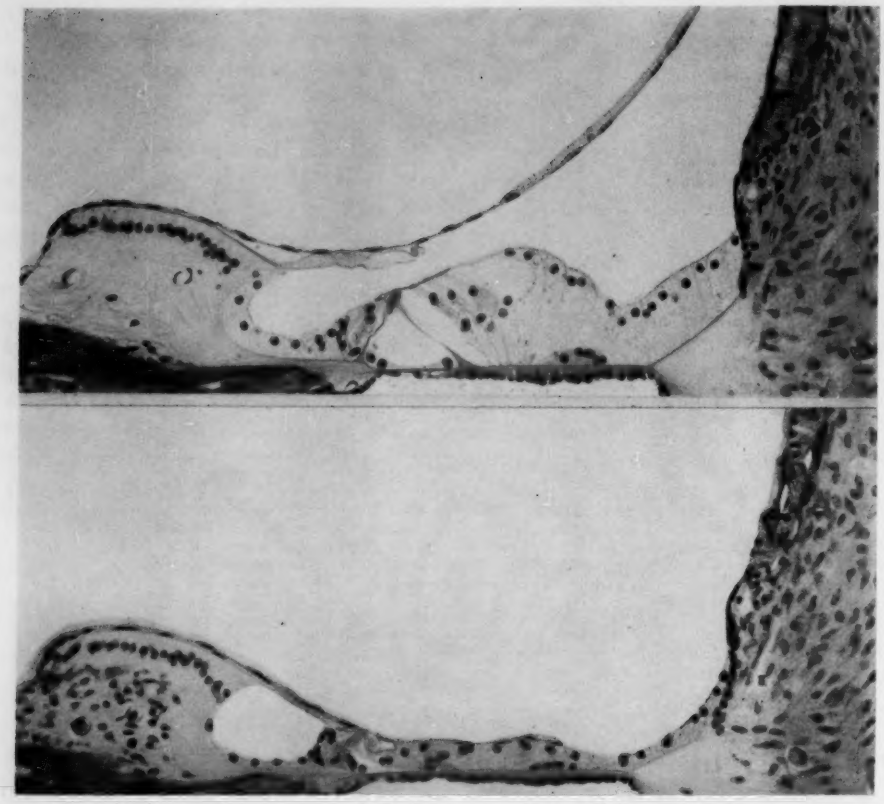


Fig. 7.—Compression of organ of Corti in a control ear. Guinea Pig D-72, twenty-one days after injection of quinine hydrochloride in right ear. Upper photomicrograph, right organ of Corti. There is absence of nuclei in substantia propria of limbus and collapse of Reissner's membrane, but the cytoarchitecture of organ of Corti is preserved. Lower photomicrograph, control (left) organ of Corti. All characteristics of compression are present. Hematoxylin and eosin; × 250.

organ of Corti according to the histologic description given by Wittmaack [4] and Mygind.[5] The assessment showed that five quinine-injected specimens and five controls presented compression phenomenon of varying degree, from minute distortion of structures to complete collapse of organ of Corti. In two cases the phenomenon was localized in the basal turn, and in eight it was extended throughout the cochlea. Figure 6 illustrates a case in which the quinine-injected ear shows compression, while the control side does not. Figure 7 illustrates the reverse case, i. e., the control side only shows compression.

Reissner's membrane. The position of Reissner's membrane for quinine-injected and control ears is summarized in the Table.

Position of Reissner's Membrane in Control and Injected Ears

	Standard	Convex	Collapsed Over Limbus	Collapsed Over Organ of Corti	Broken & Collapsed
Controls	14	0	5	2	3
Injected	6	2	8	3	5

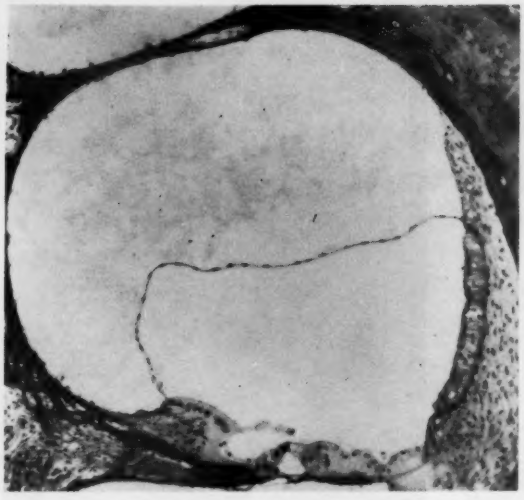


Fig. 8.—Dilatation of cochlear duct. Guinea Pig D-124, twenty-four hours after injection of quinine sulfate in the right ear. Reissner's membrane bulged into scala vestibuli throughout the cochlea. Degeneration of the external hair cells. Other structures showed no abnormalities. Notice that there is no compression of the organ of Corti even though the cochlear duct is dilated. Hematoxylin and eosin; × 100.

The standard position includes straight membranes and those with small displacement. Other findings, whether localized to some region or extending throughout the cochlea, were classified as the columns in the Table indicate. Comparison of the control and quinine-injected ears showed that the latter presented a larger incidence of collapse of Reissner's membrane.

Dilatation of the cochlear duct was found in two quinine-injected ears (Fig. 8).

Primary cochlear neurons. The observation on nervous tissue was limited because of the histologic technique. Hematoxylin and eosin staining is useful for estimation of gross abnormalities but not for fine details. Nevertheless, two observations are worth mentioning: firstly, diminution of cell bodies localized to the first turn of the cochlea in several chronic preparations; secondly, lack of staining of nerve fibers in spiral lamina, associated with disintegration of the organ of Corti.

3. Changes in the Vestibule: Collapse of the membranous walls of the vestibule was observed more frequently in quinine-injected than control ears. The collapse may be localized, or it may involve the entire system.

It was surprising to find that 17 specimens out of 24 presented no histologic damage to the vestibular receptors. The other seven specimens presented lesions in the vestibular organ as follows. In two ears, all the sensory epithelium was disintegrated by a suppurative labyrinthitis. In one ear, vestibule and semicircular canals were totally occupied by bony tissue. The other four ears showed degenerative changes in sensory epithelium, mainly in the macula saccularis (Figs. 9 and 10). The preponderance of lesions in the macula saccularis may be due to direct impact of the poison upon the receptor rather than sensitivity factors.

C. Cochlear Responses.—1. Acute preparations: Injection of quinine hydrochloride or sulfate into the middle ear or application of a drop on the round window modified cochlear responses in a few minutes. Sensitivity of both cochlear microphonics (CM) and action potentials (AP) decreased rapidly. In 12 minutes AP were barely visible and amplitude of CM reduced to about 50% of the reference level. These findings are illustrated in Figure 11. Both CM and AP may remain depressed for several hours, but eventually partial or full recovery was observed. A case of early recovery of cochlear potentials is illustrated in Figure 12.

2. Chronic Experiments: Most of the animals tested five or more days after in-

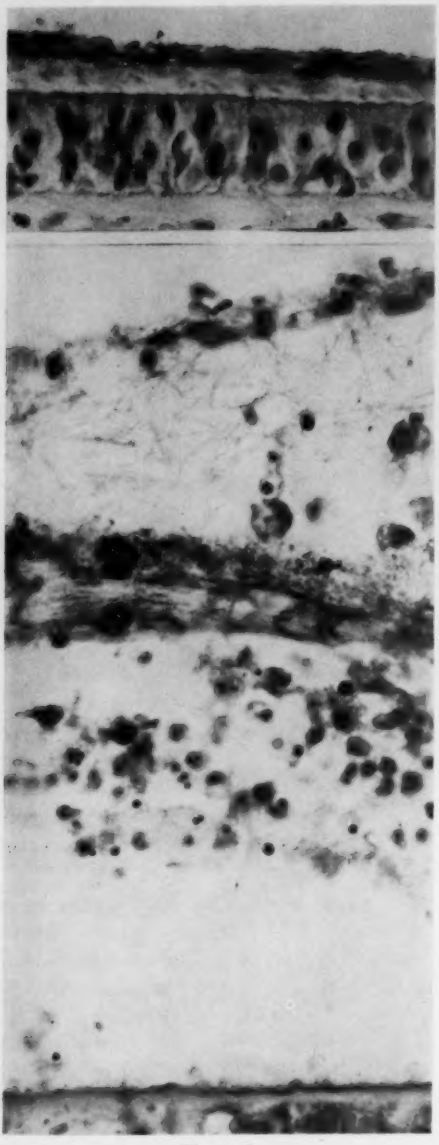


Figure 9

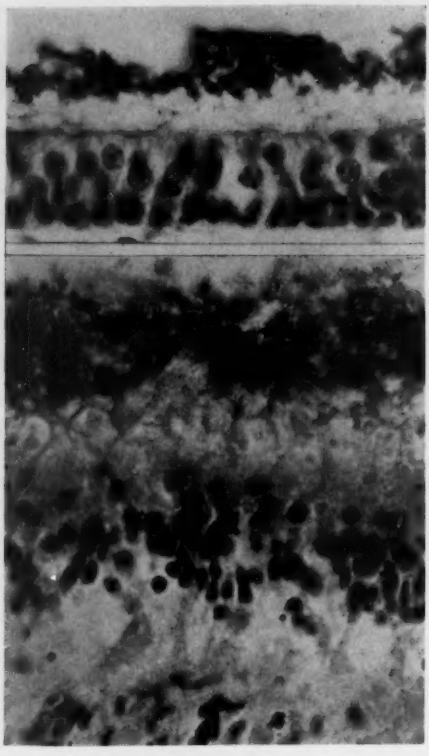


Figure 10

Fig. 9.—Disintegration of macula saccularis. Guinea Pig D-52, two days after injection of quinine hydrochloride in the right ear. The upper photomicrograph shows a section of the macula saccularis of the control ear. The lower photomicrograph shows a section of the quinine-injected ear. The sensory epithelium was detached and cells were disintegrated. Detachment and swelling produced increase in height of the macula. Hematoxylin and eosin; × 700.

Fig. 10.—Disintegration of macula utricularis. Guinea Pig D-47, two days after injection of quinine sulfate in right ear. The upper photomicrograph is a section of control ear, and the lower one is a section of the quinine-injected one. In the latter, sensory epithelium was detached and cells are in the process of disintegration. Hematoxylin and eosin; × 700.

jection of quinine showed no cochlear responses to any frequency. Microscopic observation of temporal bones showed some damage in sensory epithelium of first turn and/or conductive mechanism, as already described. In a few animals, however, the cochlear responses were present but both thresholds and maximum voltage were depressed. The specimens of these animals showed degenerative changes of sensory organ in the first turn and some damage in the conductive mechanism.

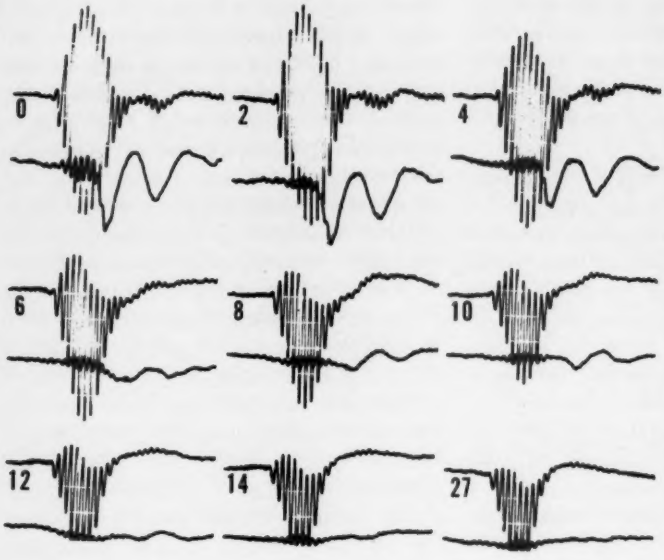


Fig. 11.—Effect of an aqueous solution of quinine hydrochloride on cochlear potentials. The numerals indicate time in minutes after a drop of the solution was put on the round window. Repression of cochlear responses progressed rapidly. In 12 minutes AP was barely visible and the amplitude of CM was reduced to about 50% of the reference level (Record 0). The negative shift of CM is due to persistence of strong negative summating potential. Stimulus, voltage, and time calibrations are shown in Figure 12.

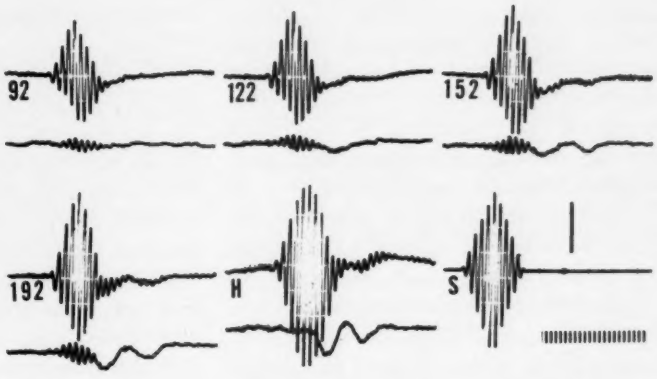


Fig. 12. — Recovery of cochlear potentials after application of an aqueous solution of quinine hydrochloride on the round window. These records belonged to the animal illustrated in Figure 11. The numerals represent time in minutes after application of quinine. Record H, which shows a substantial recovery of both CM and AP was taken 20 hours after the application. Record S, stimulus 8,000 cps tone pips. Voltage calibration, 200 microvolts. Time calibration, 10,000 cps.

Comment

The vestibular signs which appeared in the first few minutes after injection of quinine into the middle ear were characteristic of unilateral labyrinthectomy. In the case of quinine hydrochloride, the effect must be produced by the drug because it was dissolved in distilled water, and injection of distilled water alone did not produce vestibular signs at all. In case of quinine sulfate, probably there was a combined action of quinine and alcohol, because when alcohol was injected alone it reproduced the vestibular signs. Absence of histologic damage in vestibular receptors in many animals and disappearance of vestibular signs in a few hours indicate that the receptors were temporarily paralyzed. Postural nystagmus and asymmetry in rotatory reactions, seen for a few days after injection of quinine, represent a persistent vestibular disturbance. The asymmetry in rotatory reactions clearly indicates that at least the horizontal semicircular canal was involved. The postural nystagmus was of paroxysmal type, and the question of whether semicircular canals or utriculus, or both, are responsible for postural nystagmus still remains to be answered.

These observations suggest that either administration of a large dose of quinine or an idiosyncrasy may produce functional vestibular disturbances, but a permanent lesion in the sensory epithelium is not likely to be produced.

The results of our method in the cochlear cell population support the findings of Covell,[8,9] i. e., external hair cells and stria vascularis are the structures most sensitive to quinine. To these we may add the limbus. Severity of the lesion depends on amount of drug which penetrates through the round window and on the distance it may travel along the cochlea. Fibrosis, new bone formation, disintegration of organ of Corti, detachment, and necrosis of stria vascularis (generally localized to first 4 mm. of cochlear duct) indicate that quinine was concentrated in this region. Spread and dilution along the cochlear leads to damage of most sensitive cells, i. e., limbus, external hair cells, and stria vascularis. In both limbus and external hair cells, the lesion was permanent. Edema, globular formation, and vasodilation observed in the stria vascularis of acute preparations were temporary, because these changes were not seen in specimens of animals killed five days after injection. Detachment seemed to be the process which leads to permanent damage of the stria vascularis.

The incidence of hypotonic degeneration was equal in quinine-injected and control ears, which suggests that the phenomenon could be an artifact. This suggestion, however, has several objections. 1. The quinine-injected ears have a tendency for Reissner's membrane to collapse, which is in agreement with the theory of Mygind [5] about the mechanism of the phenomenon. 2. Compression in the control ears may be due to a secondary effect after absorption of quinine in the ear into which it was injected. 3. There is evidence that quinine may reach the control ear, which obviously can be accomplished through Eustachian tubes. The evidence is new bone formation in the tympanic cavity of control ears in the absence of infection or lesions which may explain it. Therefore, compression in control ears may be due to passage of quinine from one ear to the other. 4. We failed in the past [7] to confirm the theory of Mygind,[5] i. e., intravital perfusion may lead to compression of the organ of Corti. Furthermore, our experience for several years with a large number of animals showed that intravital perfusion only exceptionally is associated with compression of organ of Corti. In the series reported here, the animals were fixed with intravital perfusion. However, this is the first time we found a large incidence of compression phenomenon. This suggests that this incidence may be related to administration of quinine and consequently supports the opinion of Wittmaack.[4]

The question whether quinine may affect the vestibular and/or cochlear nuclei and their primary neurons remained to be investigated with more appropriate histologic methods.

Recapitulating, these experiments support the opinion of Covell [8,9] that nature of the ototoxicity of quinine consists in damage to external hair cells and stria vascularis. The opinion of Wittmaack [4] that quinine produces compression of the organ of Corti, however, can not be ruled out. It is possible that, under certain conditions, quinine may induce the picture of compression, but this does not mean that, in all circumstances, the phenomenon is of vital origin. In a previous study on human temporal bones,[6] evidence was presented which indicates that compression in these cases represents an artifact rather than a vital phenomenon. Since the origin and nature of compression still remain an obscure subject, we believe more investigation in this line should be done. This is an important problem in studies of human temporal bones, where a decision must be made between artifact and vital phenomenon.

It is obvious that impairment of cochlear responses in chronic animals was due to a combined damage of sensory organ and conductive mechanism. The impairment, which

closely followed application of quinine over the round window, clearly indicates it was due to damage of the sensory organ and consequently the hearing loss was of perceptive type. The recovery of cochlear potentials observed in acute preparations was attributed to the fact that our differential electrodes were placed above the region of severe damage, that is, the region near the round window. At the location of the electrodes the cells of the organ of Corti were affected only temporarily.

Our results confirmed the findings of Rüedi et al.[3] on systemic administration of quinine. Both sets of data showed that the lesion was localized preferentially in the first turn of the cochlea. Furthermore, the two methods of quinine administration produced about the same degenerative changes in the organ of Corti.

Summary

Quinine injected into the middle ear of guinea pigs produced signs of temporary unilateral labyrinthectomy, followed by postural nystagmus and asymmetry in rotatory reactions for a few days. Some damage may be found in vestibular receptors of acute and chronic preparations.

The cochlear responses in acute preparations were impaired temporarily. The histologic studies demonstrated damage in external hair cells, limbus and stria vascularis. This lesion was particularly severe near the round window, where the organ of Corti may become necrotic.

The cochlear responses in chronic preparations were either absent or impaired. Extensive damage in conductive mechanism and degeneration of external hair cells in the basal turn were found. Near the round window, the organ of Corti and stria vascularis may be absent.

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Indications for a Submucous Resection

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When the submucous resection of the nasal septum was originated, in the early 20th century, it was advised for every deviated septum regardless of symptoms. With the discovery that allergy could cause nasal obstruction, general opinion went to the other extreme and the submucous resection went into an eclipse. This situation exists to a large extent to the present day. Even otolaryngologists are guilty of advising patients that symptoms could not originate from a badly deviated septum and that they should never agree to septal surgery. Therefore, it is thought worth while to compare the indications and the results of a series of submucous resections in order to clarify the indications.

The commonest significant deviation of the septum is a vomerocartilaginous ridge. A typical vomerocartilaginous ridge will be described. This deflection begins at the maxillary crest, inferiorly and anteriorly on one side. The deviation extends obliquely in a posterior and superior direction along the junction of the quadrilateral cartilage and the vomer. The cartilage in its inferior part is luxated from its normal position atop the vomer to one side and is elongated or overgrown. The deviated edge of cartilage overlaps the vomer along its entire length and constitutes the crest and superior part of the vomerocartilaginous ridge (Fig. 1). The maxillary crest and the superior part of the vomer are thickened and also form a longitudinal ridge. Thus the cartilage constitutes the superior part and the vomer constitutes the inferior part of the ridge. Normally, the bone (ethmoid and vomer) posterior to the cartilage is a thin plate. The posterior extension of the ridge is a spur in this thin plate. It is this posterior spur or crest which juts into the middle meatus.

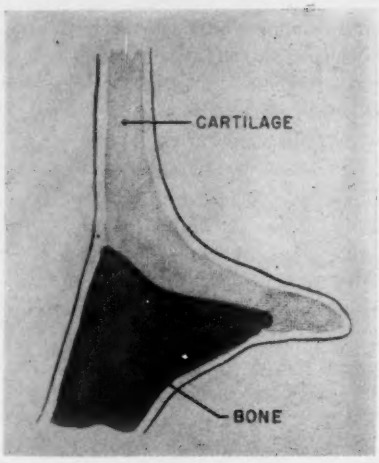
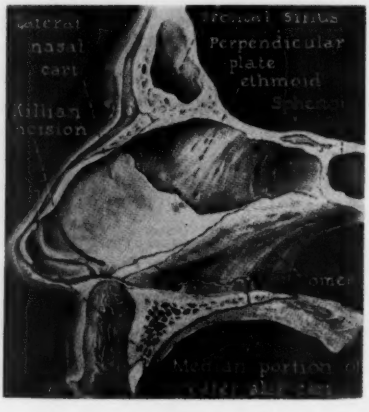


Fig. 1.—Typical formation of vomerocartilaginous ridge. Reproduced with permission from Cottle, M. H.; Loring, R. M.; Fischer, G. G., and Gaynon, I. E.: A. M. A. ARCH. OTOLARYNG. 68:301, 1958.

Fig. 2.—Lateral view of the septum. Reproduced with permission from Diseases of the Nose, Throat, and Ear, Including Bronchoscopy and Esophagoscopy, edited by C. Jackson and C. L. Jackson, Philadelphia, W. B. Saunders Company, 1945.



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Read before a staff meeting of the Department of Otorhinology, Temple University Medical Center (David Myers, M.D., Professor and Head of the Department).

The vomerocartilaginous ridge is not V-shaped with a trough or hollow on the concave side but is solid. The posterior part of the ridge does not extend all the way back to the choana but can be observed by posterior rhinoscopy. The posterior edge of the septum is in the midline. The exact etiology of the ridge is disputed. Some authors ascribe it to trauma in childhood; others, to developmental anomalies. Mosher [6] ascribed it to irregular development of the incisor teeth.

I reviewed the complaints of 100 consecutive patients, in the office, who presented a moderate to a severe deflection of the septum. All these patients had deflected septums, and all had some otolaryngologic complaint which brought them to the office. The various chronic complaints are listed (Table 1). Many patients had multiple symptoms and signs, and each symptom was included in the Table. From this short uncontrolled series, only trends may be deduced. Among the 30 patients with nasal stuffiness, allergy or vasomotor rhinitis was suspected in 24. The ear complaints included otitis media, serous otitis media, and Eustachian tube catarrh.

It can be seen that postnasal discharge, nasal stuffiness, maxillary sinusitis, and coryza were the most frequent, in that order.

Specific indications for a submucous resection and illustrative cases will be presented. Fischer [3] tabulated the relative frequency of indications for submucous resection in children as follows: nasal obstruction in 52%, ear complications in 15%, epistaxis in 10%, sinustitis in 9%, frequent colds in 9%, sore throats in 5%, and headaches in 4%. In my series, 38 consecutive submucous resections are analyzed. One patient underwent submucous resection merely as an approach to a posterior nasal tumor and is not included. All these patients are adults. In Table 2 each symptom or sign is listed and the effect of the submucous resection is noted. Since each patient had multiple complaints, the total number of symptoms adds up to more than 38. Inclusion in the improvement column indicates that the benefit is at least sufficient to make the operation worth while to the patient. Otherwise the case is listed as a failure.

TABLE 1.—*Review of Symptoms in One Hundred Patients with Deviated Septums*

Symptom	Patients, No.
Postnasal discharge	46
Nasal stuffiness	30
Maxillary sinusitis	26
Frequent coryza	22
Ear complaints	12
Headaches or facial pains	10
Cough	10
Nasal polyps	8
Sore throats	4
Epistaxis	4

TABLE 2.—*Results Obtained After Thirty-Eight Submucous Resections*

Complaint	Improvement	Failure	Total
Nasal obstruction	14	4	18
Headaches	4	6	10
Frequent respiratory infections	6	2	8
Post nasal discharge	5	2	7
Maxillary sinusitis	4	1	5
Allergy	1	3	4
Epistaxis	4		4
Chronic ulcer of septum	1		1

1. *Sinusitis.* — The vomerocartilaginous ridge may obstruct the exit from a maxillary or ethmoid sinus and lead to infection. With a history of chronic or recurrent maxillary sinusitis, in particular, it is helpful to correct the deviation. Sinus surgery may be performed at the same time.

Maxillary sinusitis was benefited in four of my five patients. The one failure has recurrent bilateral maxillary and ethmoid sinusitis and advanced rheumatic heart disease. A left Caldwell-Luc operation only helped partly. His attacks and complications are much less severe than formerly, but I believe this is due to early treatment and prompt antral lavage. Antibiotics never helped.

An illustrative case is presented:

A 54-year-old woman gave the typical history of purulent discharge of a chronic left maxillary sinusitis for many years. At my initial examina-

tion, on Feb. 14, 1958, she also had a left otitis media, which cleared with treatment. She was very nervous and refused antral washes. Roentgenograms of the sinuses confirmed the diagnosis of a left maxillary sinusitis. The septum had a left vomerocartilaginous ridge which blocked the left middle meatus. Various antibiotics and nasal cleansing in extensive trials produced no improvement. On April 14, 1958, a left Caldwell-Luc operation, combined with a submucous resection, was done. She did well thereafter. On Oct. 3, 1958, she was treated for an infection of the upper respiratory tract, which cleared with medications and nasal cleansing.

The other patients listed only had a submucous resection.

2. *Nasal Obstruction.*—It is this group which must be differentiated from allergy or vasomotor rhinitis. Where the latter conditions play the chief role the patient will complain of nasal blockage mostly on the side opposite to the deviation. With undisputed allergy most physicians try to improve the patient by allergic measures first. For the vasomotor rhinitis, treatment consists of nasal cleansing, reduction in carbohydrate intake, avoidance of irritating fumes and tobacco, and treatment of hypothyroidism.

Where the conservative approach does not improve the patient in a matter of a few months, a submucous resection should be advised. The submucous resection improves the function of the nose so that allergic and medical treatment can better benefit the patient. One must assure the patient that surgical intervention will not cure him but he will require other measures.

Among the 18 patients in this series with nasal obstruction five submucous resections failed. Of these five patients, three have a definite allergy. Management by a competent allergist had failed because of exposure to an allergen at their work. In my past experience, those patients with severe allergy and asthma never were improved by a submucous resection, and no such case is included here. One patient whose sole complaint was nasal obstruction received no benefit from the submucous resection, nor did he benefit from extensive medical and allergic care. His original deviation of the septum was only moderate, and the final diagnosis may be psychogenic. The remaining 13 patients received worth-while benefit. One of these patients also had a synechia, which was removed, and another patient had a nasal polyp removed. Two other patients appeared allergic but refused allergic studies. Both received sufficient improvement from a submucous resection, and so they refused further treatment.

Illustrative cases follow:

A 50-year-old woman had been treated since 1943 for repeated colds, sore throats, and nasal stuffiness. Allergy tests had disclosed a 2+ intradermal reaction to house dust, wool, and coffee. Roentgenograms of the paranasal sinuses were negative. She worked at textiles and refused to give up this job. Local nasal medications, treatments by an allergist, electrocautery of the inferior turbinates, oral antihistamines, and oral ephedrine had not helped. House-dust vaccine had been of no avail in the past. She had visited physicians at weekly intervals for years. Complete otolaryngologic examination revealed the patient's condition normal except for the septum, which had a right vomerocartilaginous ridge and a left spur. In July, 1956, a submucous resection was performed. She was well until November, 1956, when exposure to paint brought on coryza. Again in June and March, 1957, she had coryza attacks. A telephone call in January, 1959, disclosed that she was well. Her only complaint was moderate dryness of the nose.

A 22-year-old man was first seen in August, 1957, for nasal blockage, epistaxis, and recurrent septal hematomas. He was a boxer, and each bout caused a septal hematoma and epistaxis. The external nose deviated to the right slightly, and there was a mild cartilaginous saddle of the dorsum. The septal cartilage was deviated markedly to the left, but it was also widened and split. Because of these complications he agreed to abandon boxing. He refused a rhinoplasty. On Dec. 26, 1957, a submucous resection was performed with use of the Goldman technique. A telephone call in December, 1958, disclosed that he had no complaints.

3. *Frequent or Persistent Infections of Upper Respiratory Tract.*—A deviated septum can prolong an infection of the upper respiratory tract. A patient may believe that the deviation does not cause any trouble, but close questioning will often reveal an increased incidence or prolongation of nasal infections. There were eight patients in this group, of whom six reported improvements from the submucous resection. However, a

much longer follow-up is necessary to substantiate this.

Two patients with chronic otitis media and repeated respiratory infections were included in this group. Both underwent mastoidectomy and later a submucous resection, and have had excellent results.

An illustrative case follows:

A 20-year-old girl presented a history of frequent and repeated upper respiratory infections since early childhood. She had discharging ears until 1947. The right side of the nasal septum displayed a vomerocartilaginous ridge, but caudally the septum deviated to the left. An allergy survey, which included 40 of the commoner nasal allergens, elicited negative results. The remainder of the otolaryngologic examination, including rotengenograms of the paranasal sinuses, was negative except for purulent exudate in each nasal passage. On March 18, 1958, a submucous resection was performed through a right-sided Killian incision. Since that time trouble has been negligible. A telephone call in January, 1959, disclosed that she had not visited a physician since the submucous resection, and had no complaints.

4. *Postnasal Discharge or Frequent Sore Throats.*—Postnasal discharge is ascribed to increased flow, thickened mucus, or stasis of the mucous flow. The deviation can alter nasal physiology and stimulate the mucous glands. Most postnasal discharge is not due to a deflection of the septum, and a course of conservative therapy should be tried first. Seven patients complained of postnasal discharge. This included one woman whose sole complaint was postnasal discharge. The five improved patients reported partial but not complete disappearance of their postnasal discharge. One patient with repeated sore throats reported maximal improvement.

5. *Epistaxis.*—Repeated epistaxis from the septum is usually associated with a deviation. The bleeding commonly arises from a concave area of the septum and not from a convexity. If epistaxis is not cured by cauterization, a submucous resection will often effect a cure. Apparently the resultant fibrosis obliterates the bleeders.

An illustrative case follows:

A 57-year-old woman gave a history of repeated epistaxis especially on the right side for many years. Medical treatments had not helped. At the initial treatment, on Dec. 1, 1956, electrocautery was used for an active bleeder. The septum was deviated considerably to the right side. Hypertension was present. The otolaryngologic examination, roentgenograms of the sinuses, and routine blood studies were normal. In January, 1957, a submucous resection was performed. Bleeding was relatively profuse during the operation, and nasal packs were required for four days postoperatively. Her subsequent course was excellent, and there has been no further bleeding.

Most epistaxis is on a local nasal basis and most of these arise from the septum. Occasionally one encounters epistaxis due to a systemic disease. The following such case is presented because of its unusual features:

A 45-year-old man had been treated for hepatic cirrhosis for several years. He manifested left epistaxis in early 1956. In March, 1957, severe epistaxis required hospitalization for two weeks elsewhere. Intensive medical treatment, blood transfusions, and continuous nasal packing were administered. In April, silver nitrate was applied to the septum by his generalist. On May 11 and 15 I used electrocautery for recurrent epistaxis from various places on the septum. On May 16 he was hospitalized again, and on that date his red blood cell count was 2,860,000; hemoglobin, 8.5 gm. %; cephalin flocculation test, 4+; prothrombin concentration, 80%, and Wassermann, negative. An internist gave intensive medical care for the cirrhosis plus blood transfusions. Because of the poor progress, on June 4 I did a submucous resection. During the operation bleeding was moderate, but the friable mucosa melted away under manipulation and a large perforation was sustained. Packing postoperatively was required until June 10. He was discharged from the hospital on June 22. Purulent crusting in the nasal passages, particularly at the edges of the perforation, continued for many months. This necessitated weekly nasal cleansing and the daily use of a Nichols Nasal Siphon irrigation unit at home. Occasionally a mild bloody ooze started spontaneously from the edges of the perforation. Absorbable gelatin sponge (Gelfoam) rarely helped, and petrolatum gauze packing was usually required. He was treated by me until June 6, 1958, at which time the nose appeared very clean. He is still under the care of the internist, but his nasal troubles are cleared. There has been no further bleeding.

It is possible that this patient may have cleared on medical treatment alone, but the submucous resection seemed to hasten control of the hemorrhage.

With systemic disease or a cauterized septum a perforation is a definite danger.

6. *Headaches or Facial Pains.*—It is this category which has given the poorest results, and yet it is this indication which impels most referrals from general practioners or internists. No patient in this series who had headaches as the sole complaint received complete relief. There were 10 patients who had headaches as a prominent complaint. All four who were improved had occasional headaches afterward, but the headaches were less frequent and less severe. Those patients whose sole complaint was headache without other otolaryngologic complaints such as nasal obstruction, frequent URI, or postnasal discharge, uniformly had failures. One patient operated on in 1955 has had complete relief from headaches since then.

7. *Chronic Ulcer of the Septum.*—One patient with this condition is presented:

A 51-year-old woman complained of itching, crusting, and bleeding in the right nasal passage since 1936. Repeated local treatments had only helped temporarily. The septum was straight, but there was a large chronic ulcer at Kiessilback's area on the right side. Electrocautery on two occasions helped temporarily for weeks. The patient had a morbid fear that cancer would develop. On Feb. 19, 1958, a localized submucous resection and biopsy of the area were performed. Subsequently the crusting ceased. The biopsy specimen showed chronic inflammatory tissue.

Technique

Only a few parts of the operation will be described. One may approach the correction through a Killian incision, hemitransfixion, full transfixion, or a Cottle approach. The Killian incision was used in this series except where a caudal deflection was present. The Killian incision was a vertical incision about 2 cm. in length on the cutaneous side of the mucocutaneous junction. The incision was made on the same side as the ridge. The radical resections performed in former years are inadvisable. However, fracturing over deviated portions had failed in my hands. It is only necessary to remove those portions which obstruct. I have never been able to reset the overlapping piece of cartilage into a groove on the vomer, and I resect this overlapping cartilage. The bony ridge on the vomer is chiseled away; the posterior bony ridge is excised, and the underlying plate of bone is so thin it breaks away along with this ridge. This part of the ridge, far posteriorly in the nose, is the part which obstructs the sinus orifices and must be removed. Finally the maxillary crest is chiseled away. Occasionally there is hemorrhage from the anterior palatine vessel, so it is best to do the maxillary crest area last.

It is explained to the patient that he will be hospitalized for one to two days. Operation is always done under local anesthesia, except in children. There is no pain after adequate infiltration of procaine. Postoperatively, petrolatum gauze nasal packing is inserted for one day, but if there is no oozing during operation it is feasible to eliminate any packing. The patient is warned that the nose will be stuffy for a week, as if he has a cold, but there will be no pain.

The commonest complication noted after a submucous resection is dryness of the nose. This seemed commoner in the older age group and where mucosa was lacerated. In none of these patients was it severe. It is not a contraindication to the operation.

Summary and Conclusions

A submucous resection is indicated where the septal deviation is associated with nasal obstruction, suppurative sinusitis, repeated upper respiratory infections, epistaxis from the septum, and chronic ulcer of the septum.

The vomerocartilaginous ridge is the commonest septal deflection which requires correction.

Often a submucous resection is accessory, and other measures are also necessary.

Headaches or facial pains can seldom be ascribed to a deflected septum.

The submucous resection technique should be mastered so that it becomes a relatively minor operation.

There has been overemphasis on conservatism as to correction of a deviated septum.

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Audiology Section

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An Exploratory Investigation of Delayed Progressive Neural Hypacusis in Children

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Introduction

Attempts to classify early deafness in children are necessarily complicated by the factors of immaturity and communication. Problems of conductive loss have been relatively easy to diagnose and treat since the discovery of antibiotics and antihistamine therapy for infection and allergic control. However, the availability of drugs provides a great temptation to the practitioner to overlook the possibility of neural involvement. Indeed, nerve deafness in children is much commoner than suspected, and only recently have testing techniques been developed with sufficient precision to differentiate conductive from neural loss.

Within the area of nerve deafness in young children, the clinician is faced with baffling etiological considerations. If the problem is detected within a few months after birth, the probability of congenital and/or hereditary etiology is great, but with time such a diagnosis becomes less tenable, especially with no readily discernible external and/or causal factors. The fact remains that apparently there is a segment of the pediatric-otologic population which exhibits normal auditory acuity at birth but develops a neural type loss in early childhood. The loss generally becomes evident between the ages of 3 and 6 years and apparently increases with age. That the loss is basically neural in origin is supported by common otologic differential diagnostic techniques, which show no air-bone gap and Rinne-positive tuning-fork response.

The incidence of young patients with this type of loss was sufficiently high in my clinical practice for me to undertake an exploratory systematic analysis of case histories and bibliographic search for supporting data. This is, therefore, an exploratory investigation of children with delayed degenerative neural hearing loss. The following questions were proposed for consideration pertinent to the problem:

1. Is there any ordered and/or chronological deterioration of hearing?
2. Do serial pure-tone audiograms exhibit any definite pattern of loss by frequency?
3. Will the data yield any predictive evidence on hearing loss for speech?

Review of the Literature

A search into literature relating to delayed neural deafness revealed little clinical material of importance to this particular investigation. The large majority of studies reviewed were concerned either with etiological factors per se or with discussions of histological data. Altman (1950)[1] has well stated the dilemma faced by such investigators as Steurer (1922, 1926),[2-4] who tried to resolve problems of etiology based on

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histological findings, "In many instances a clear cut distinction between these types (acquired and congenital) is much easier in theory than in practice, especially when the findings are limited to the membranous parts of the cochlea."

Politzer (1903)[5] described the syndrome, noting the delayed onset and progressive deterioration, but stated that the loss was restricted to high frequencies. Alexander (1927)[6] felt that this type of deafness was acquired, but he also felt that the loss could possibly remain stationary and not regress. He also theorized that the loss revealed a congenitohereditary etiology. Albrecht (1922, 1940)[7,8] described cases of "partial nerve deafness" in which familial patterns of transmission varied, but such transmitted losses were apparently noticeable in early infancy. Dolowitz' monumental study (1950)[9] of Mormon records apparently discovered no cases of a delayed, neural, hereditary type. Bordley and Hardy (1951),[10] in a study of 296 children with neural loss, isolated 82 cases with a delayed onset during early childhood—all acquired and secondary to infection. There were no serial studies available on those cases. A careful review of the "Clarke School Studies Concerning the Heredity of Deafness," by Hopkins and Guilder (1949),[11] showed no case type of pedigree similar to the population here studied.

Johnsen's study (1954)[12] on 111 Danish school children, though pertinent, described transmitted losses as a high-tone type, with three separate loss types based on slope characteristics. Losses, though equally bilateral, were asymmetric and not progressive. A careful review of the investigation did not show that hearing was essentially normal up to early childhood and hence described a condition unlike the one investigated herewith.

In summary, a review of literature pertinent to delayed, apparently transmitted, neural type of hearing loss in children failed to uncover information of direct importance to this study. Therefore, a contribution on delayed neural hypacusis in children might well be significant.

Material

Medical records and audiograms were available in my office files. It was estimated that about 25 fit criteria essential to the study. Basic criteria forced subjects into two major classifications: (1) those children for whom normal pure-tone thresholds were available and (2) those children for whom no initial normal audiogram was available but hearsay evidence was fairly reliable. To eliminate any possible contamination with cases having conductive involvements, only those cases with an *insignificant* (less than 5 db.) or *no* air-bone gap were chosen. The basic criterion, of course, stipulated that no subject would have demonstrated clinical neural hypacusis prior to the age of 3 years. A careful screening of the cases resulted in the selection of 13 children and 1 adult. (The latter case was included because the history seemed to indicate delayed hypacusis and because she was the mother of two of the subjects.)

Procedure

Within the framework of mentioned subject criteria, pure-tone threshold audiograms were arranged on master audiograms for evaluative purposes (Table). Bone-conduction curves were eliminated by definition. In order to make audiograms readable, a maximum of four representative curves were shown, each audiogram denoting a significant change in audiometric response in octaves from 125 cps to and including 4,000 cps. This procedure affected only four audiograms (Figs. 1, 2, 10, and 13), for which more information was available but which were independently judged redundant by my two colleagues. By averaging the three middle frequencies, 500, 1,000, and 2,000 cps (commonly acknowledged as the best estimate of speech threshold from pure-tone data), an adequate speech level was derived. Such an estimate was thought to be a more operational description of hearing status. Consequently, mean variations (arithmetic averages) were computed on each subject and for each ear. Data were then massed for purposes of interpretation. The case history and other otologic data were assembled in condensed form both to support and to illustrate case findings.

Presentation of Cases

Case 1.—An 8-year-old white boy was brought to the office by his parents. The father, a physician, reported apparent normal hearing to the age of 6 years. The hearing loss was moderate and seemed to fluctuate. The child was in the third grade of

Raw Data: Speech-Frequency Pure-Tone Thresholds—All Measurements, Including Averaged Differences Between Ears

Case No.	Age	Sex	Right Ear				Left Ear				Av. Difference Between Ears
			500	1,000	2,000	Av.	500	1,000	2,000	Av.	
1	8	M	10	15	10	10	10	10	0	5	5
	9		5	15	10	7½	10	5	10	7½	0
	9½		20	25	20	20	20	20	30	20	0
	9¾		25	30	30	27½	25	20	25	22½	5
	10½		30	25	35	27½	35	40	30	32½	5
	11		65	60	55	57½	55	55	60	55	2½
	12		55	75	60	57½	65	65	60	62½	5
	13		80	75	65	70	75	75	70	72½	2½
	15		75	85	85	80	80	75	70	72½	7½
	17		80	85	75	77½	75	70	60	65	12½
2	4	M	20	20	20	20	25	15	25	20	0
	4		45	35	30	32½	35	45	50	40	7½
	4		40	40	50	40	45	45	40	42½	2½
	5		55	50	60	52½	60	55	45	50	2½
	6		85	85	80	82½	85	85	90	85	2½
	10		80	90	90	85	85	90	85	85	0
3	5	M	25	35	40	30	25	25	30	25	5
	10		35	75	75	55	25	35	65	30	25
4	6	M	20	20	20	20	20	25	15	17½	2½
	9		65	60	75	62½	20	35	100	27½	35
	11½		90	95	100	92½	95	100	100	97½	5
5	5½	M	5	5	5	5	5	0	5	2½	2½
	6½		20	35	30	25	60	65	65	62½	37½
	7		35	50	40	37½	70	60	60	60	22½
6	8	M	30	35	25	27½	25	45	55	35	7½
	12		45	60	40	42½	55	65	65	60	17½
7	6	M	35	35	65	35	35	45	60	40	5
	9		60	65	55	57½	65	70	70	67½	10
8	4	F	10	5	5	5	5	5	10	5	0
	5½		15	15	10	12½	15	10	15	12½	0
	7		30	20	20	20	15	20	30	17½	2½
	8		50	60	55	52½	50	70	80	60	7½
9	6	M	15	15	25	15	15	15	25	15	0
	7		25	25	30	25	25	25	30	25	0
	8		35	35	40	35	35	40	40	37½	2½
10	5	F	10	10	10	10	10	10	10	10	0
	5½		20	20	20	20	20	20	20	20	0
	6		20	20	20	20	20	20	20	20	0
	6½		30	30	30	30	30	30	30	30	0
	7		50	55	50	50	50	50	55	50	0
11	4	F	30	30	25	27½	25	20	25	22½	5
	5		45	45	45	45	40	40	55	40	5
	6		55	55	55	55	55	55	60	55	0
12	4	F	0	5	0	0	5	0	0	0	0
	5		30	25	35	27½	30	35	30	30	2½
	7		50	50	55	50	50	55	55	52½	2½
13	5	F	15	20	0	7½	20	10	15	12½	5
	5½		25	25	20	22½	20	25	30	22½	0
	6		35	35	40	35	25	25	30	25	10
	6½		50	55	50	50	50	60	50	50	0
	7		65	65	65	65	70	75	80	72½	7½
14	33	F	50	80	85	65	20	80	90	50	15
	36		50	80	85	65	20	80	80	50	15
	40		50	100	100	75	30	80	100	55	20

a school for normal children and had achieved average grades.

History.—The pregnancy was uneventful, and birth was normal. There was a questionable encephalitis at the age of 18 months, with no residuum except a "nervousness" that disappeared entirely by the time the child was age 4 years. There had been no exanthemata. The child suffered from nasal and bronchial allergies, which were treated successfully with antihistamines. Prior to his coming to our office, a tonsillo-adenoidectomy with radium therapy to the nasopharynx had been performed, at the age of 6 years, following symptoms which included one attack of otitis media and fluctuating hearing loss. (The mother reported that her own hearing began to regress at about the age of 17.)

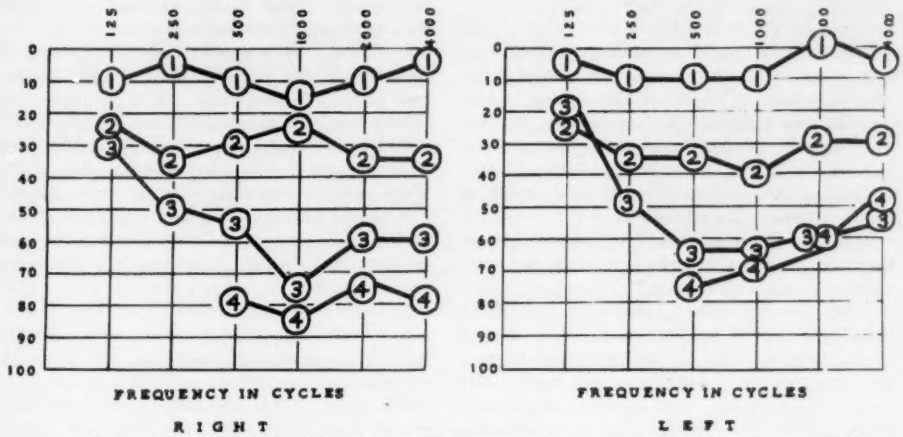

Fig. 1.—Serial pure-tone air-conduction audiograms of Case 1, taken at ages 8 (*1*), 10½ (*2*), 12 (*3*), and 17 (*4*) years.

Physical Examination.—The eardrums were found to be intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx was free of adenoid tissue, and both Eustachian tube orifices were patent. Pharyngeal tonsils had been removed completely. The general physical examination was otherwise well within the limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 1, Curve *1*, as a series of pure-tone threshold audiometry tests from October to December, 1948, shows that bone conduction and air conduction were equal, with no gap. A diagnosis of mild bilateral neural hypacusis (so called "perceptive deafness") was made, with hearing within lower normal limits. A final diagnosis of severe progressive bilateral neural hypacusis was made as serial audiometry revealed the regression (Fig. 1, Curves *2, 3,* and *4*).

Course.—Serial reexamination (11 in all) revealed a steady downward regression of auditory threshold response. Speech intelligibility deteriorated, and in October, 1951, a hearing aid was recommended. This was not obtained until the following April and has been since exchanged several times for stronger models. The general physical condition has remained good during this period of time, with no major illness or operations.

Case 2.—A 4-year-old boy was brought to the office by his parents. Their chief complaint was

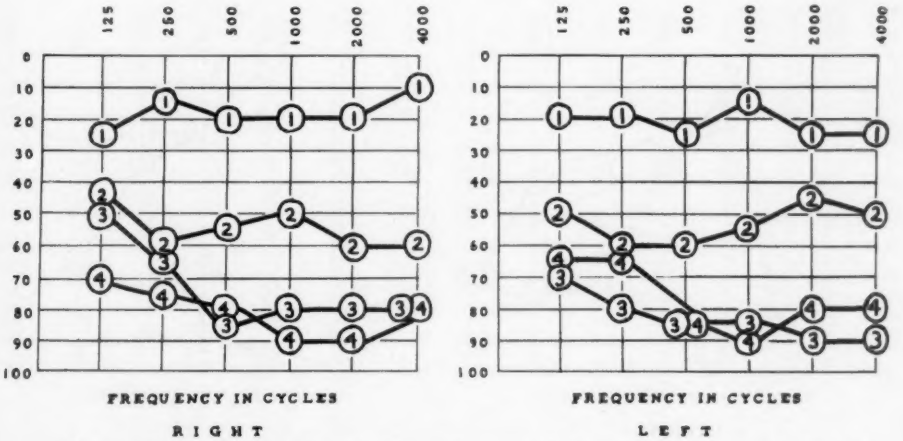

Fig. 2.—Serial pure-tone air-conduction audiograms of Case 2, taken at ages 4 (*1*), 5 (*2*), 6 (*3*), and 10 (*4*) years.

recent hearing loss. The hearing loss was only moderate but did not diminish.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. The child had a history of infrequent attacks of mild tonsilloadenoiditis. The mother reported that her own hearing began to regress at about the age of 17. An older brother had a history of hearing regression, which first appeared at the age of 5 years (Case 1, a sibling).

Physical Examination.—The eardrums were found to be intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork was Rinne positive (AC>BC). The nasopharynx had a small central adenoid, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils were moderately enlarged, with injection of the anterior pillars. The general physical examination was otherwise well within limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 2, Curve *1*, shows the pure-tone threshold audiometer test taken March 2, 1951. Bone conduction and air conduction were equal, with no gap. An initial diagnosis of bilateral neural hypacusis was made. A final diagnosis of severe progressive bilateral neural hypacusis was made as serial audiometry traced the regression (Fig. 2, Curves *2*, *3*, and *4*).

Course.—On the basis of the physical findings and the audiometric tests, recommendations were not made for a tonsilloadenoidectomy. Serial audiograms (six in all) revealed a steady downward regression. Speech intelligibility deteriorated, and a hearing aid was recommended in October, 1951. The aid was not obtained until the following April. It has been changed several times for more powerful models. The general physical condition has remained good during this period of time, with no major illness or operations.

CASE 3.—A 6½-year-old boy was brought to the office by his parents. Their chief complaint was hearing loss of two years' duration. The hearing loss was moderate and had not diminished.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. There were no other otologic or general complaints. The mother had a hearing loss (acquired) at the age of 12 years, as a result of scarlet fever, with chronic otitis media. This loss was relatively static.

Physical Examination.—The eardrums were found to be intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx had a small central adenoid, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils were moderately enlarged, with injection of the anterior pillars. The general physical examination was otherwise well within limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 3, Curve *1*, shows the pure-tone audiometric test, taken May 26, 1954. Bone conduction and air conduction were equal, with no gap. An initial diagnosis of moderate bilateral neural hypacusis was made. A final diagnosis was made of severe right and moderate left bilateral neural hypacusis (Fig. 3, Curve *2*).

Course.—In 1954, recommendations were made for lipreading instruction, continued attendance in regular school, and additional audiometric tests in three months. However, the child was not seen again until Aug. 26, 1957. During this period speech intelligibility had deteriorated and a hear-

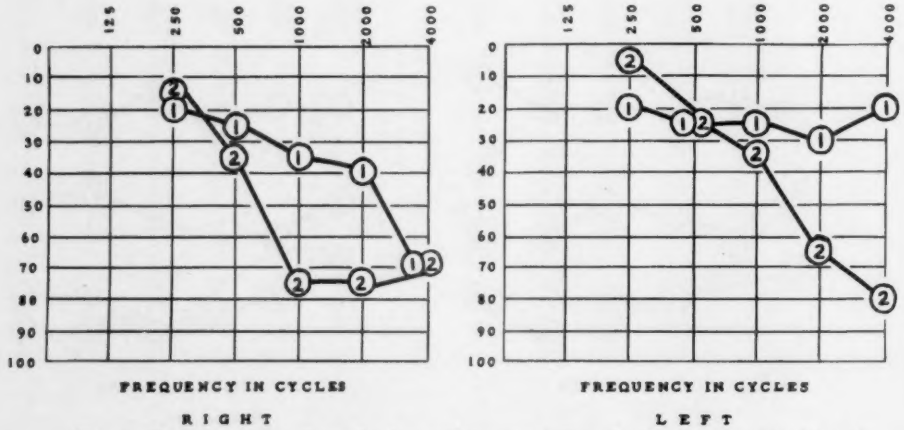

Fig. 3.—Serial pure-tone air-conduction audiograms of Case 3, taken at ages 6½ (*1*) and 10 (*2*) years.

ing aid had been purchased independently at a hearing aid "store." Amplification obtained with the aid was only moderate. The parents had brought the child to the office for the purpose of having the hearing aid checked, as they were considering the purchase of another model. The aid used had been purchased within the last six months, and, although the child's hearing had been deteriorating rather steadily in the three intervening years, the child had not been seen by an otologist.

CASE 4.—A 4½-year-old white boy was brought to the office by his parents. The chief complaint was recent hearing loss. The hearing loss was moderate and had not diminished.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. There were no other otologic or general complaints.

Physical Examination.—The eardrums were found to be intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx had a small central adenoid, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils were moderately enlarged, with injection of the anterior pillars. The general physical examination was otherwise well within limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 4, Curve *1*, shows the pure-tone audiometer test, taken on March 18, 1950. Bone conduction and air conduction were equal, with no gap. A diagnosis of moderate neural hypacusis was made. A final diagnosis of severe bilateral neural hypacusis was made as serial audiometry revealed the regression (Fig. 4, Curves *2* and *3*).

Course.—The child was not seen again until March 20, 1954, and then only at the request of a local hearing and speech clinic, where the child was receiving auditory training. There had been a steady regression in hearing and speech, and the child was attending special classes for the hard of hearing in addition to receiving lipreading instruction at the clinic. The audiogram revealed a further progression of neural loss, more pronounced in the right ear. Despite recommendations for continued testing, no further audiometric tests were done until June 17, 1957, when the child was 11½ years of age. The neural loss had progressed bilaterally to a severe neural hypacusis. Speech intelligibility was very poor, and apparently amplification did not help speech discrimination. The general physical condition had remained good during this period of time, with no major illness or operations.

CASE 5.—A 5½-year-old white boy was brought to the office by his parents. The chief complaint was recent hearing loss. The hearing loss was moderate.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. There was a chronic nasal allergy, being treated with antihistamines and elimination diet. A tonsilloadenoidectomy had been performed one year previously. There were no other otologic or general complaints.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx had no adenoid tissue, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils had been removed completely. The general physical examination was

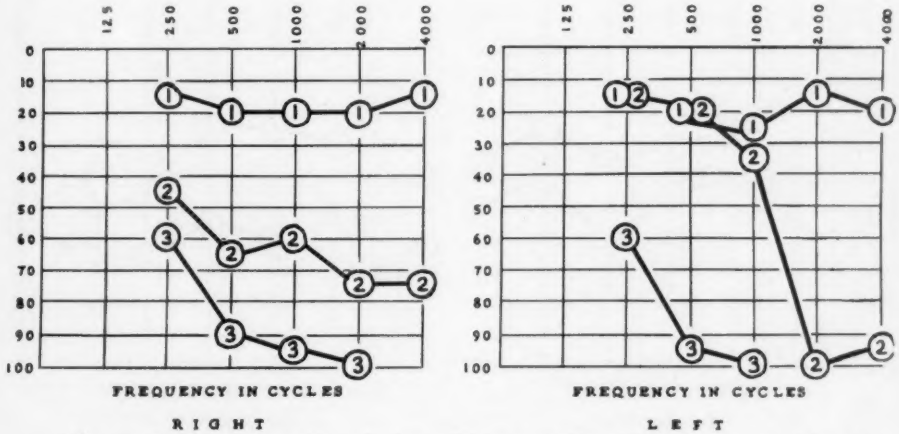

Fig. 4.—Serial pure-tone air-conduction audiograms of Case 4, taken at ages 6 (*1*), 9 (*2*), and 11½ (*3*) years.

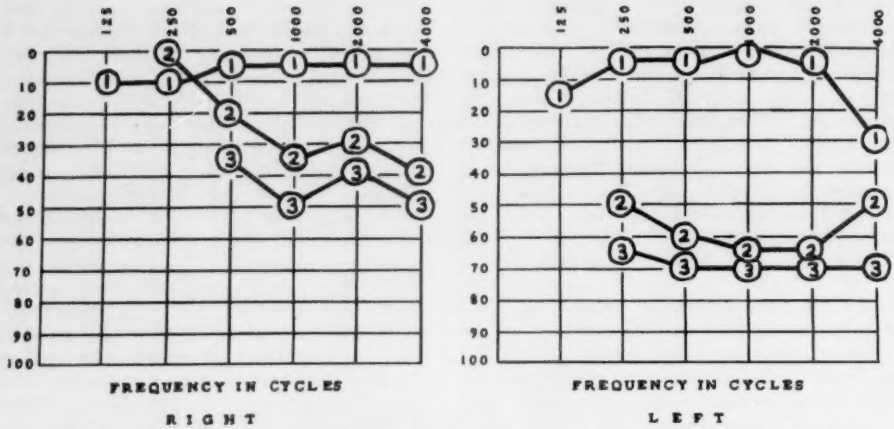

Fig. 5.—Serial pure-tone air-conduction audiograms of Case 5, taken at ages 5½ (*1*), 6½ (*2*), and 7 (*3*) years.

otherwise well within limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 5, Curve *1*, shows the pure-tone audiometer test, taken Feb. 9, 1955. Repeated tests on three separate occasions revealed normal hearing. Speech tests confirmed normal hearing. A final diagnosis of severe bilateral neural hypacusis was made as serial audiometry revealed the regression (Fig. 5, Curves *2* and *3*).

Course.—The child was to return in six months but was not seen again until March 27, 1956 (13 months later), at which time there was a deterioration by pure-tone audiometric tests and speech-reception testing. The right ear had a speech-reception-threshold (SRT) score of 15, and the left ear, a score of 50. Testing on Jan. 16, 1957, revealed further deterioration, with an average SRT score of 38 on the right and 63 on the left. The speech had deteriorated somewhat, and the child was wearing a hearing aid and receiving lipreading instruction.

CASE 6.—An 8½-year-old white boy was brought to the office by his parents. The chief complaint was recent hearing loss. The hearing loss was moderate and seemed to be progressive.

History.—The pregnancy was uneventful, and birth was normal. The patient had had chicken pox and measles while of nursery school age. No note was made of hearing loss after the exanthemata. Audiometric tests which had been made several years previously at school were reported as normal. Unfortunately, however, these audiograms were unavailable. Hearing loss had become

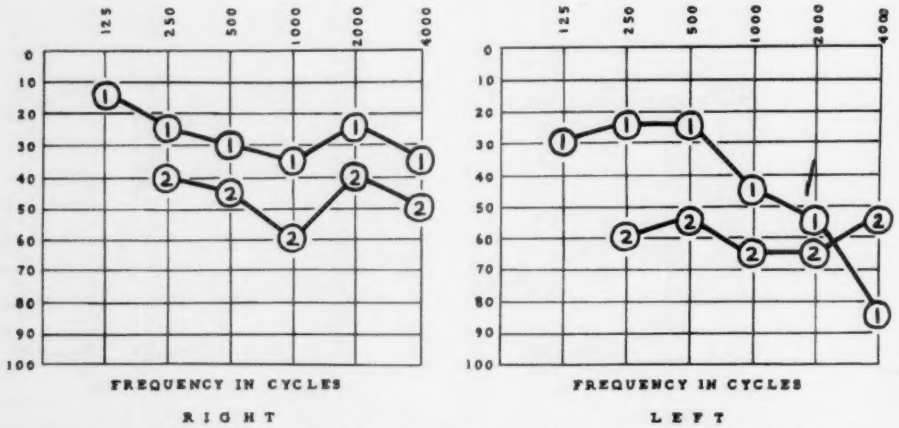

Fig. 6.—Serial pure-tone air-conduction audiograms of Case 6, taken at ages 8 (*1*) and 12 (*2*) years.

more apparent within the past six months and was confirmed by audiometric testing done at school and at a local hearing and speech clinic during this time. There were no other otologic or general complaints.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx had a small central adenoid, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils were moderately enlarged, with injection of the anterior pillars. The general physical examination was otherwise well within limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 6, Curve *1,* shows the pure-tone audiometric test, taken on Nov. 9, 1953. Bone conduction and air conduction were equal, with no air-bone gap. A diagnosis of bilateral moderate neural hypacusis was made. A final diagnosis of bilateral severe neural hypacusis was made as serial audiometry traced the regression (Fig. 6, Curve *2*).

Course.—A hearing aid, as well as lipreading instruction, was recommended. Audiometric tests were repeated serially through August, 1957 (including speech-reception-threshold tests), during which time the hearing regressed progressively and bilaterally. The general physical condition has remained good during this period of time, with no major illness or operations.

CASE 7.—A 6-year-old white boy was brought to the office by his parents. The chief complaint was recent hearing loss, more pronounced within the past six months. The hearing loss was moderate but becoming more severe.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. A tonsilloadenoidectomy had been performed at the age of 4 years. There were no other otologic or physical complaints.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. A tuning-fork test was Rinne positive (AC>BC). The nasopharynx was clear of adenoid tissue, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils had been removed completely. The general physical examination was otherwise well within the limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 7, Curve *1,* shows the pure-tone audiometric test taken March 22, 1954. Bone conduction and air conduction were equal, with no air-bone gap. A diagnosis of moderate neural hypacusis was made. A final diagnosis of severe bilateral neural hypacusis was made as serial audiometry revealed regression (Fig. 7, Curve *2*).

Course.—A hearing aid was recommended and obtained. Lipreading instruction was also initiated. Audiometric tests (including speech-reception-threshold tests) were repeated serially, through Aug. 12, 1958. The hearing regressed progressively and bilaterally. The general physical condition has remained good during this period of time, with no major illness or operations.

CASE 8.—A 4½-year-old white girl was brought to the office by her parents. The chief complaint was recent hearing loss. The hearing loss was slight.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. There were no other otologic or general complaints.

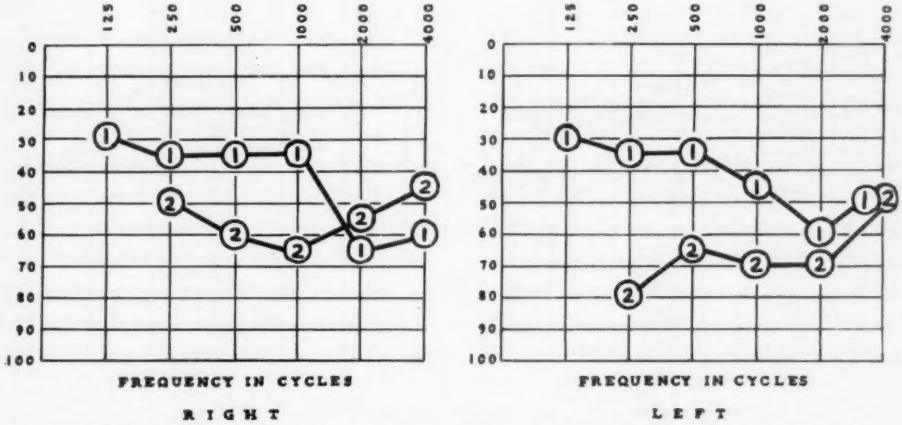

Fig. 7.—Serial pure-tone air-conduction audiograms of Case 7, taken at ages 6 (*1*) and 9 (*2*) years.

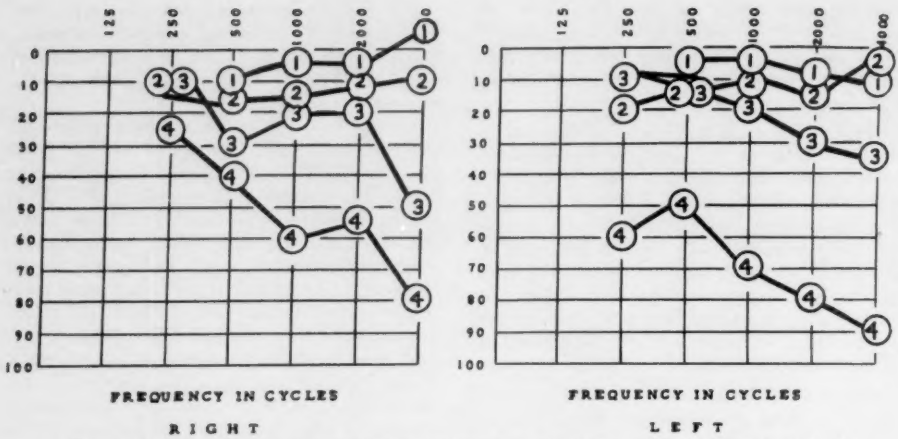

Fig. 8.—Serial pure-tone air-conduction audiograms of Case 8, taken at ages 4 (*1*), 5½ (*2*), 7 (*3*), and 8 (*4*) years.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx had a small central adenoid, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils were moderately enlarged, with no injection of the anterior pillars. The general physical examination was otherwise well within the limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 8, Curve *1*, shows the pure-tone audiometric test, taken Jan. 10, 1955. Bone conduction and air conduction were equal, with no air-bone gap. A diagnosis of normal hearing was made, with the recommendation for repeated studies. However, a final diagnosis of severe bilateral neural hypacusis was made as serial audiometry revealed the regression (Fig. 8, Curves *2*, *3*, and *4*).

Course.—Audiometric tests revealed progressive hearing loss and deteriorating speech. Lipreading was recommended in July, 1957, and a hearing aid, in September, 1957. The child attended a school for the hard of hearing. The general physical condition has remained good during this period of time, with no major illness or operations.

CASE 9.—A 6-year-old white boy was brought to the office by his parents. Their chief complaint was recent hearing loss. The hearing loss was slight but progressive.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. A tonsilloadenoidectomy had been performed at

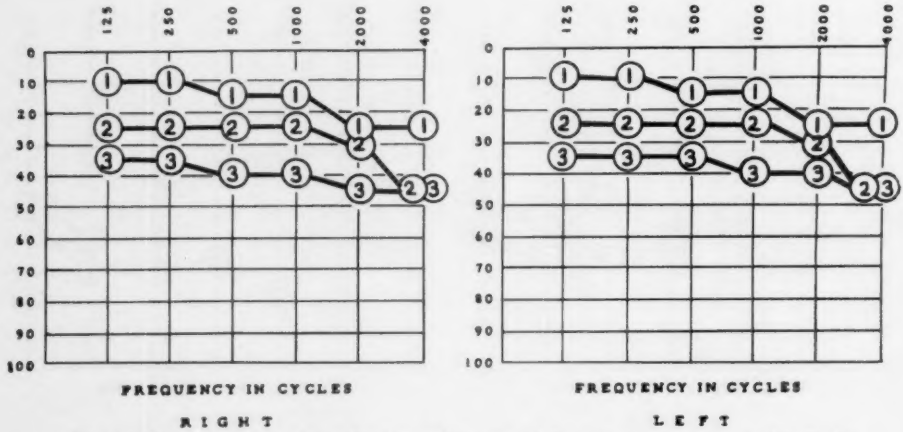

Fig. 9.—Serial pure-tone air-conduction audiograms of Case 9, taken at ages 6 (*1*), 7 (*2*), and 8 (*3*) years.

the age of 5 years. There were no other otologic or general complaints.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx was clear of adenoid tissue, and the Eustachian tube orifices were nonobstructed. Pharyngeal tonsils had been removed completely. The general physical examination was otherwise well within the limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 9, Curve *1*, shows the pure-tone audiometric test, taken July 10, 1956. Bone conduction and air conduction were equal, with no air-bone gap. A diagnosis of moderate neural hypacusis was made. A final diagnosis of bilateral severe neural hypacusis was made as serial audiometry revealed the regression (Fig. 9, Curves *2* and *3*).

Course.—Audiometric tests revealed more progressive loss of hearing. Speech deteriorated to a moderate extent. Lipreading was recommended in May, 1957; a hearing aid, in June, 1958. The child is attending a regular school and using a hearing aid. The general physical condition has remained good during this period of time, with no major illness or operations.

CASE 10.—A 5-year-old white girl was brought to the office by her parents. The chief complaint was recent hearing loss. Hearing loss was slight and seemed progressive.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. There were no other otologic or general complaints.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx had a small central adenoid, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils were moderately enlarged, with no injection of the anterior pillars. The general physical examination was otherwise well within the limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 10, Curve *1*, shows the pure-tone audiometer test, taken on Jan. 8, 1956. Bone conduction and air conduction were equal, with no air-bone gap. A diagnosis of normal hearing was made, with the suggestion that the child be seen and retested in three months. A final diagnosis of moderately severe bilateral neural hypacusis was made as serial audiometry revealed the regression of hearing function (Fig. 10, Curves *2*, *3*, and *4*).

Course.—Hearing steadily regressed. Lipreading and a hearing aid were recommended in January, 1957. The child is attending a school for the hard of hearing. The general physical condition has remained good, with no major illness or operations.

CASE 11.—A 4-year-old white girl was brought to the office by her parents. The chief complaint was recent hearing loss. The hearing loss was moderate and showed progression.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. There were no other otologic or general complaints.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The

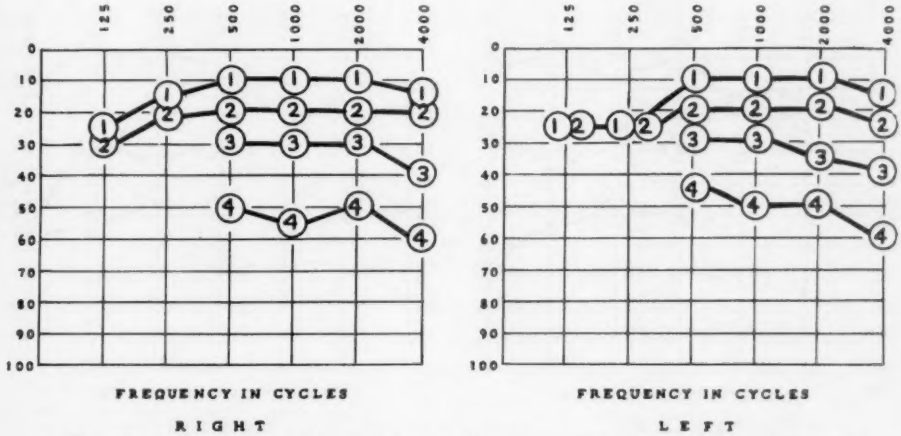

Fig. 10.—Serial pure-tone air-conduction audiograms of Case 10, taken at ages 5½ (*1*), 6 (*2*), 6½ (*3*), and 7½ (*4*) years.

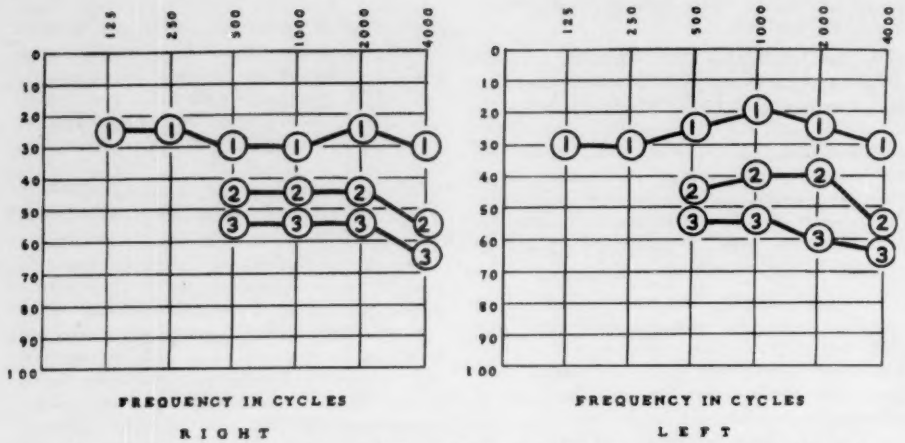

Fig. 11.—Serial pure-tone air-conduction audiograms of Case 11, taken at ages 4 (*1*), 4½ (*2*), and 5 (*3*) years.

tuning-fork test was Rinne positive (AC>BC). The nasopharynx had a small central adenoid, and the Eustachian orifices were not obstructed. Pharyngeal tonsils were moderately enlarged, with injection of the anterior pillars. The general physical examination was otherwise well within limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 11, Curve *1*, shows the pure-tone audiometric test, taken April 8, 1956. Bone conduction and air conduction are equal, with no air-bone gap. A diagnosis of moderate neural hypacusis was made. A final diagnosis of severe bilateral neural hypacusis was made as serial audiometry traced the regression (Fig. 11, Curves *2* and *3*).

Course.—After the initial audiometric test, a recommendation for lipreading instruction and serial audiometry was made. As audiometry and speech testing revealed a decline in hearing acuity, the further recommendation of a hearing aid was made, in January, 1957. Speech has deteriorated, and the child is attending a school for the hard of hearing. The general physical condition has remained good, with no major illness or operations.

Case 12.—A 4-year-old white girl was brought to the office by her parents. The chief complaint was recent hearing loss. The hearing loss was slight but seemed progressive.

History.—The pregnancy was uneventful, and birth was normal. There had been no exanthemata. There were no other otologic or general complaints.

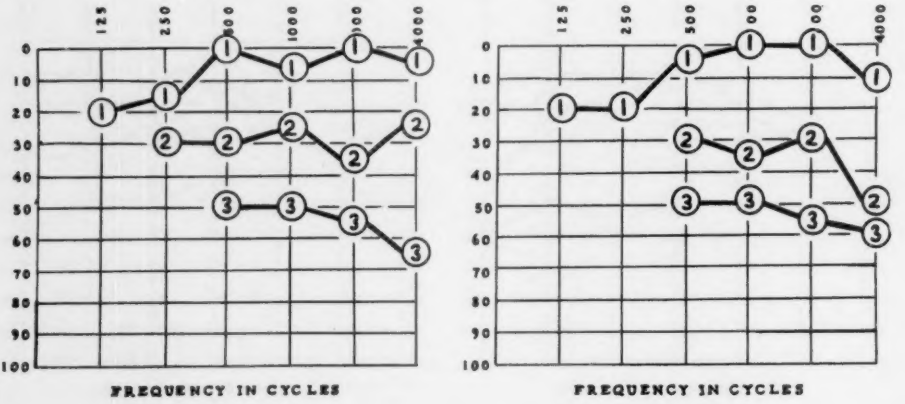

Fig. 12.—Serial pure-tone air-conduction audiograms of Case 12, taken at ages 4 (*1*), 5 (*2*), and 7 (*3*) years.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx had a small central adenoid, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils were moderately enlarged, with injection of the anterior pillars. The general physical examination was otherwise well within the limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 12, Curve *1*, shows the pure-tone audiometric test, taken in October, 1956. Bone conduction and air conduction were equal, with no air-bone gap. A diagnosis of normal hearing was made. A final diagnosis of severe bilateral neural hypacusis was made as serial audiometry traced the regression.

Course.—As audiometric tests were repeated serially, the recommendation for lipreading instruction was made in September, 1957, and for a hearing aid, in May, 1958. The child is attending a school for the hard of hearing. The general physical condition has remained good, and there has been no major illness or operations.

CASE 13.—A 5-year-old white girl was brought to the office by her parents. The chief complaint was recent hearing loss. The hearing loss was moderate but seemed to be progressive.

History.—The pregnancy was uneventful, and birth was normal. She had had chicken pox, with unilateral right otitis media, at the age of 4 years; this had responded well to antibiotics. A tonsilloadenoidectomy had also been performed in her fourth year. There were no other otologic or general complaints.

Physical Examination.—The eardrums were intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx was clear of adenoid tissue, and the Eustachian tube orifices were not obstructed. Pharyngeal tonsils had been removed completely. The general physical examination was otherwise well within the limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 13, Curve *1*, shows the pure-tone audiometric test, taken May 8, 1956. A diagnosis of moderate neural hypacusis was made. A final diagnosis of severe bilateral neural hypacusis was made as serial audiometry traced the regression.

Course.—Lipreading instruction was recommended in January, 1957, and a hearing aid, in December, 1957. The child has entered a school for the hard of hearing. The general physical condition has remained good, and there has been no major illness or operations.

CASE 14.*—A 33-year-old white woman presented herself to the office with a complaint of hearing loss of 15 years' duration. The hearing loss had been progressive.

History.—The pregnancy was uneventful, and birth was normal. She had had childhood exanthemata, with no residual. There had been a tonsilloadenoidectomy in childhood. Her pregnancies had

* Case 14 is presented but is not included as statistical material in the section on Results and Discussion. It was felt that, inasmuch as Case 14 was the mother of Cases 1 and 2 and reported a history of progressive neural loss, such a presentation might well be an exhibit of a logical sequela to delayed neural hypacusis.

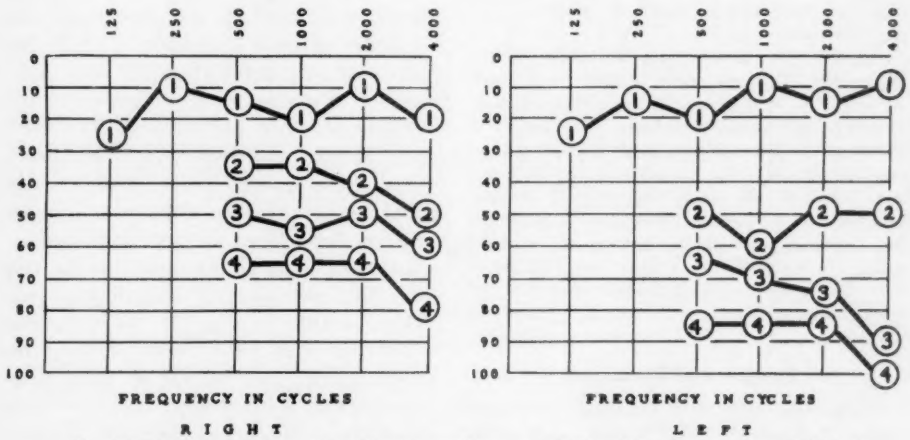

Fig. 13.—Serial pure-tone air-conduction audiograms of Case 13, taken at ages 5 (*1*), 6 (*2*), 7 (*3*), and 7½ (*4*) years.

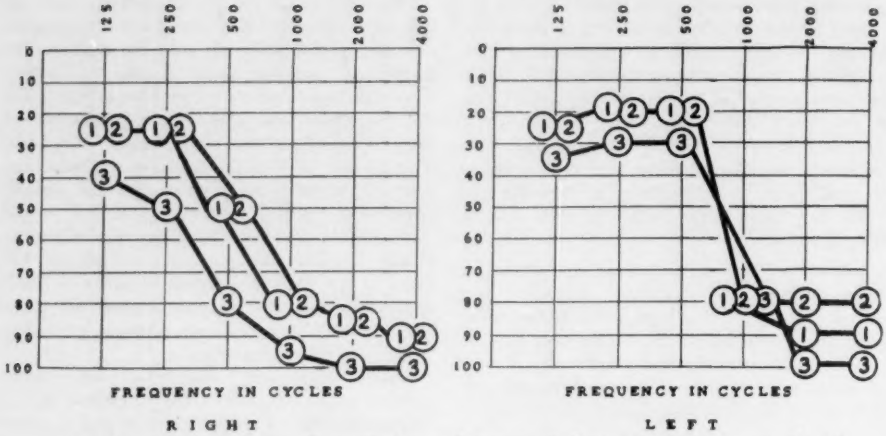

Fig. 14.—Serial pure-tone air-conduction audiograms of Case 14, taken at ages 33 (*1*), 36 (*2*), and 40 (*3*) years.

not affected her hearing. Of her three children, two have a history of progressive hearing losses.

Physical Examination.—The eardrums were found to be intact and translucent; they moved well on pneumatic otoscopy and inflated easily on politzerization. The tuning-fork test was Rinne positive (AC>BC). The nasopharynx was clear of adenoid tissue, and the Eustachian tube orifices were patent. Pharyngeal tonsils had been removed completely. The general physical examination was otherwise well within limits of normality and noncontributory to the otologic findings.

Audiometric Findings and Diagnosis.—Figure 14, Curve *1*, shows the pure-tone-threshold audiometric test, taken Oct. 25, 1948. Bone conduction and air conduction were equal, with no air-bone gap. An initial diagnosis of bilateral neural hypacusis was made. A final diagnosis of severe bilateral neural hypacusis was made as serial audiometry traced the regression.

Course.—The patient had originally refused audiometry "because her hearing was good enough." As hearing regression evidenced itself in two of her children, she reluctantly agreed to be tested. Audiometric tests were made at various intervals (though not as often as we desired) and showed a slow steady regression of hearing, primarily in the higher frequencies. The patient had volitionally acquired a hearing aid in 1953, which she replaced with a more powerful aid in 1955. The general physical condition has remained good during this time, with no major illness or operations.

Results and Comment

From the raw data (Table), averaged speech-frequency (500, 1,000, and 2,000 cps) thresholds were computed in accordance with standard interpolative procedures for all cases. An inspection of these data showed that the sample population fell into two groups. (Case 14, an adult, was not included in group data.)

Five of the patients had verifiably *normal* initial speech-frequency thresholds. The other eight had *reportedly normal* hearing up to about the age of 3 years, followed by diminution of hearing acuity. If both groups assumed similar graphic characteristics, it could then be reasonably assumed that both groups were members of the same clinical population.

Data were treated in two ways. One method was based on the assumption that the better ear was a suitable index for an optimal hearing status of the patient. However, to obtain a more complete picture, averages for each ear were in turn averaged, to give a binaural speech-frequency index. Both measures could then be compared, to see whether differences, if any, between ears played an important role.

In order to trace the course of hearing loss, data were massed according to age. When plotted on a graph, the parameter would therefore be a curve showing the progression of the loss for all cases. Figure 15 shows the better-ear curve for those cases whose initial and verifiable auditory thresholds were within the normal range.

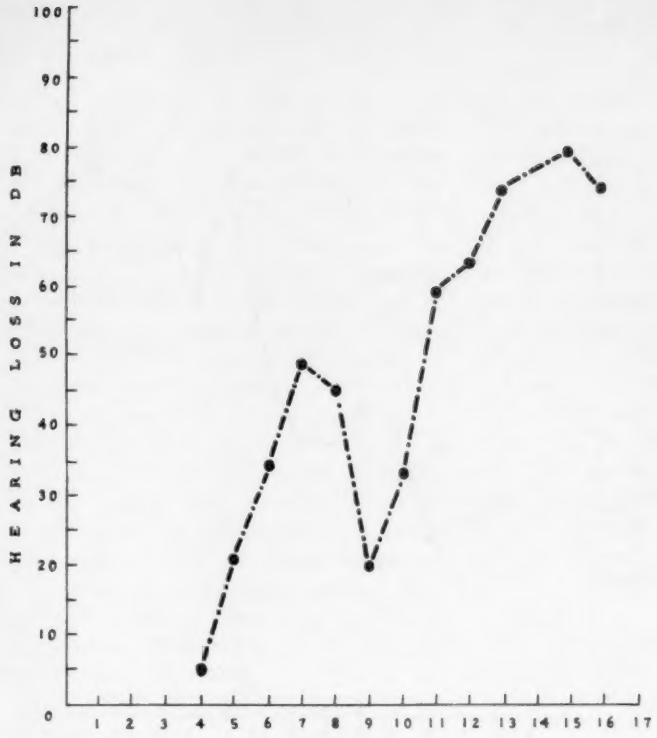

Fig. 15.—Averaged speech frequency puretone thresholds as a function of age for the better ear on five cases tested initially as normal.

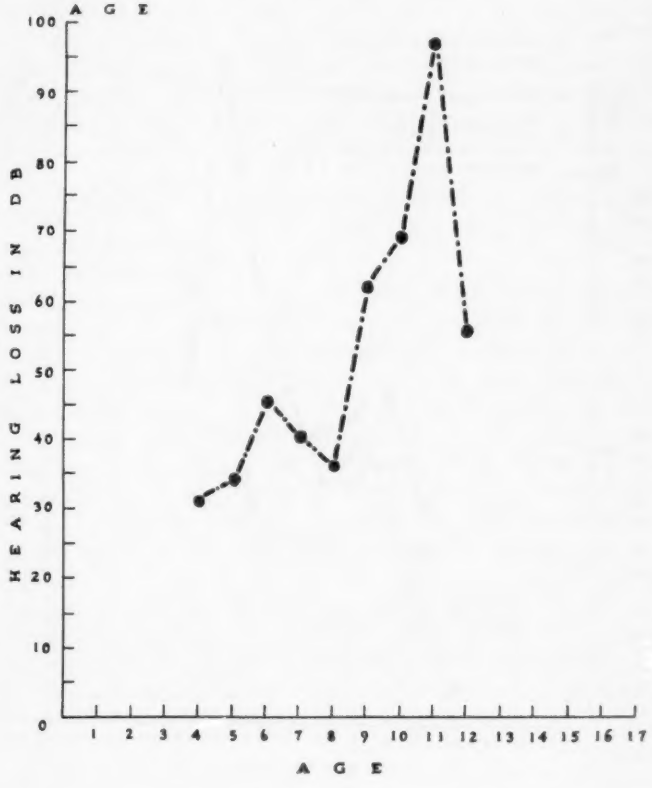

Fig. 16.—Averaged speech frequency puretone thresholds as a function of age for the better ear on eight cases with unconfirmed initial normality.

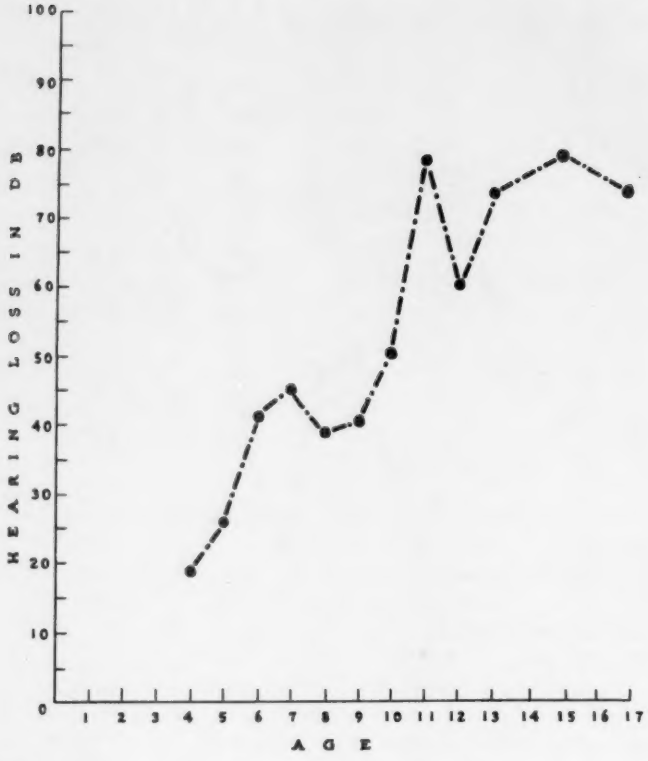

Fig. 17.—Averaged speech frequency pure-tone thresholds as a function of age for both ears on all cases (13) reported.

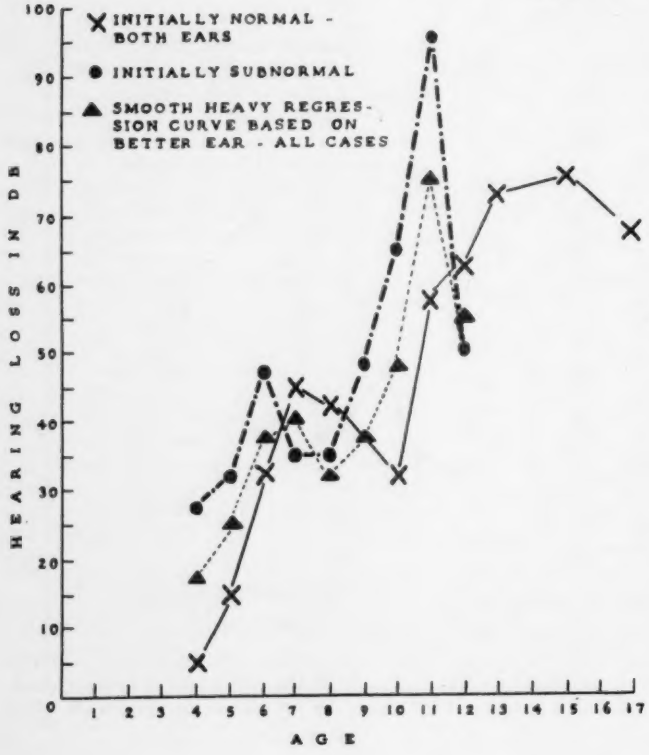
INITIALLY NORMAL - BOTH EARS
INITIALLY SUBNORMAL
SMOOTH HEAVY REGRESSION CURVE BASED ON BETTER EAR - ALL CASES
HEARING LOSS IN DB
AGE

Fig. 18.—Three curves showing slope relationships between better ear and moth ears for averaged speech frequency pure-tone thresholds as a function of age.

Although the curve generally tended to approach a regression type, a drop occurred between the ages of 7 and 10 years. Whether this is a true function or not could not be determined from such a small sample.

Figure 16, plotted from better-ear data on patients who were reported to have had normal hearing, showed the same type of dip in this age range, confirming this possibly remissive period. A drop at age 12 years was due to a relatively low threshold of 48 db. for Case 8, whose poorer ear was actually at 62 db.

If Figure 15 is superimposed on Figure 16, the trend is generally upward, tending to confirm the hypothesis that both groups were members of the same population. The "unconfirmed" group seemed to have generally higher thresholds but actually by computation showed less than a 10 db. difference on the average.

When both ears were averaged for all 13 cases, the curve tended to flatten out (Fig. 17) at around 11 years but still demonstrated a plateau at around 7 years. This might possibly indicate that the course of the loss was not regular. On the other hand, a certain amount of clinical testing error was to be expected; consequently the plateau might be attributed to chance variation with this group of patients.

Figure 18 is a summary graph, presented to demonstrate the upward and generally regressive nature of the disorder. Despite the irregularities, one cannot deny the degenerative aspect of this type of hearing loss. It is also obvious by inspection of the graphs and raw data that the loss was not *equally* bilateral nor was the course *equally* progressive, but losses did tend toward equal bilaterality. It was also interesting to note that in this sample only two cases (Cases 8 and 13) showed greater losses at 4,000 cps than at 2,000 cps. As all of the graphs were based on those frequencies most important to speech function, trends suggest the paramount factor of loss of communicative efficiency. Undoubtedly, a larger population would demonstrate that acoustic amplification and remedial procedures are necessarily an important recommendation for these patients at an early age.

Of less importance perhaps, in this exploratory investigation, was the question of sex distribution. As there were only five girls in the study, compared with eight boys, no conclusive evidence was presented to demonstrate real differences in the course of auditory deterioration. Inspection of audiograms seemed to indicate no difference between the sexes by type or frequency slope. The fact that girls were presented obviously rules out the factor of sex differences being present. Just what proportion is to be expected in the estimated clinical population may only be surmised to be similar to other neural type losses.

Case 14 was presented more as a congenitohereditary implication than as a member of the population of this investigation. As already stated in the Presentation of Cases, this patient was the mother of Cases 1 and 2. She reported her hearing loss as beginning around the age of 17. One can only guess from clinical experience that the actual onset of the loss must have occurred earlier. However, her loss at age 33 in the speech-frequency range could not be termed significantly greater than that of her children at adolescence, despite differences in total frequency-slope characteristics.

This could mean that the children inherited a degenerative type of loss but considerably earlier—as evidenced by their audiograms. As this constellation represented such a statistically unverifiable population, only guesses can be made concerning hereditary and/or genetic transmission of this form of loss. One might hazard a hypothesis that those factors might demonstrate some "snowball" effect, producing severer and accelerated losses in the offspring. Unfortunately this was the only available family group, but further investigation might show that such a familial linkage may well be a contributing factor.

Another equally tenable hypothesis might be that, prior to the age of 3 or 4 years, losses could conceivably be termed subclinical. However, until more precise means of determining auditory thresholds of infants is available to the clinician, this problem will remain unsolved.

Summary

Serial audiograms of 13 children, 8 male and 5 female, with a verified delayed form of neural hypacusis were analyzed for possible similarity and loss progression. A review of the literature seemed to indicate that such progressive losses had been noted but not systematically and serially analyzed. Case histories were presented, including a family constellation of a mother and her two children. It was decided that data extracted from speech frequencies would prove to define operationally the loss and ensuing progression. Data were therefore treated on an averaged basis, analyzed, and discussed.

Conclusions

Bearing in mind that the population in this study was small and consequently attempts to generalize on the findings were necessarily tentative, the following conclusions appear warranted:

1. Apparently there is a steady deterioration of hearing of the neural type in these cases in the speech frequencies from age 4 to about 7 years. At this age, approximately, there seems to be a leveling-off for several years, then a steady rise to 60-70 db.

2. There was no definite pattern of audiometric response by frequency. However, losses in the speech frequencies increased in a fairly flat distribution. Inspection of all frequencies showed a tendency to drop in the high end but not significantly so.

3. From the data secured in this investigation, one can safely predict that this type of hearing loss for speech increases steadily with age. Clinical observations revealed that acoustic amplification and auditory rehabilitation are a necessity.

4. Male and female subjects apparently demonstrate fairly equal acoustic regression or deterioration.

5. There is a possibility that heredity may be an important etiological factor.

6. Exploratory investigation tended to support a diagnosis of delayed progressive neural hypacusis for this type of hearing loss.

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Structural Analysis of Word Responses Made by Hard of Hearing Subjects on a Discrimination Test

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I. Introduction

Discrimination of the sounds of speech is a topic which continues to occupy the attention of investigators who are interested in nonclinical as well as clinical aspects of speech and hearing. In a report to a CHABA Symposium on Military Audiometry, Hirsh [1] pointed out that there are dimensions other than intensity required for the understanding of speech, that involve complex discriminations between frequencies, groups of frequencies, simultaneous intensities, and tonal complexities. He further states that, since all of these factors are not yet understood, perception of speech can be predicted only by testing it directly.

Numerous efforts have been made to construct tests of sound discrimination. In 1931, Travis [2] and Rasmus constructed a speech-sound discrimination test. The test items were nonsense syllables. In 1943, Templin [3] reported a test of sound discrimination which consisted of 70 pairs of consonants combined with various vowels. Norms were established for Grades 2 through 6. During World War II, the Psycho-Acoustic Laboratory at Harvard University developed the PB-50 word lists described by Egan.[4] In 1952, Hirsh and others reported a modification they made of the original PB-50's, which resulted in a smaller test vocabulary that was more rigidly phonetically balanced and which presented each word spoken only once in different positions throughout the four 50-word test lists. Holding constant the utterance of each of the words was accomplished by transcription recording. The test is referred to as C. I. D. Auditory Test W-22. It is this test which was administered to the subjects of the present study.[5] In 1950, Mansur [6] created a picture type test of sound discrimination similar to that of Travis and Rasmus. In 1953, Pronovost and Dumbleton [7] reported research which involved a revision of the Mansur test. In 1958, Schiefelbusch and Lindsey [8] reported a new picture test of sound discrimination which was structured so as to determine the ability of children to discriminate speech units as they hear them from others, as they produce them themselves, and as they evaluate them silently. Some of the tests not only demand discrimination but articulatory performance.

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II. Present Research

The purpose of the present study, however, is to relate the performance of hard of hearing subjects on a sound-discrimination task to other audiometric measures and to analyze error responses in terms of phonetic structure.

The study represents a preliminary effort and is divided into two parts. Part I deals with results of testing 400 ears of male subjects who came to the Speech and Hearing Clinic at Ohio State University for audiological evaluation. Part II deals with 110 of the original 400 ears. Fifty-five were characterized by conductive loss and fifty-five by a loss other than conductive. Classifications of conductive and nonconductive were made on the basis of otological examinations.

III. Equipment and Procedure

The audiometric tests were administered in a sound-treated room which has an ambient noise

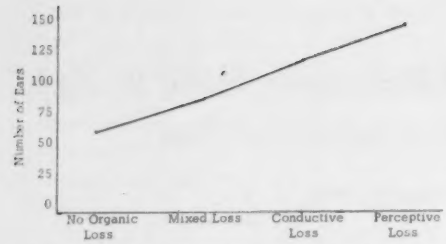

Fig. 1.—Otological diagnosis of the 400 ears.

level of 45-55 db., as measured with a General Radio Sound Level Meter set on the C-Scale. The tests were administered with an Allison 20-A audiometer through headphones. Presentation was monaural. As indicated previously, the discrimination task involved recognition of the C. I. D. Auditory Test W-22 (Lists 1-A, 2-A, 3-A, and 4-A). Level at which the lists were delivered was 40 db. above obtained Speech Reception Thresholds or at the upper limit of the audiometer in the event that the obtained SRT was so high as to preclude presentation of the lists 40 db. above it.

IV. Questions

Questions proposed at the outset of the study were as follows:

1. How does the relative frequency of occurrence of insertions, omissions, and substitutions of vowels and consonants compare?
2. Do the types of consonant confusions differ greatly for the two hearing-loss categories?
3. Do the two-, three-, four-, and five-sound words in the PB lists differ in their intelligibility?
4. Are sound omissions and insertions in error responses more frequently found in initial or final positions?
5. Does discrimination loss decrease on second presentation of the test lists?
6. Is there a difference in the frequency of repetition of the same error response by conductives and nonconductives as the test lists are presented a second time?

V. Part I. Results and Analysis

Figure 1 presents the otological diagnoses assigned each of the 400 ears. It is of interest to note that the most frequent diagnostic labels assigned were conductive and perceptive losses. Functional losses make up 15% of the total; mixed, 21%; conductive, 30%, and perceptive, 34%.

Figure 2 presents the distribution of the ears according to age range. Ages varied

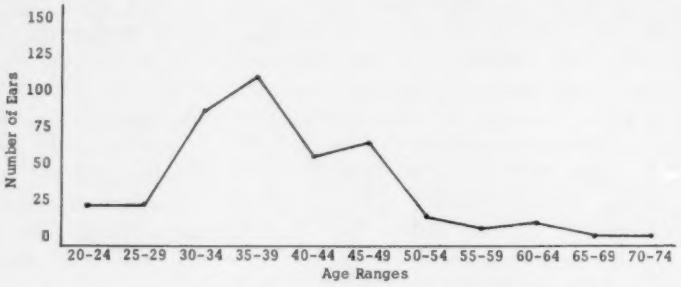

Fig. 2.—Distribution of 400 ears according to age range.

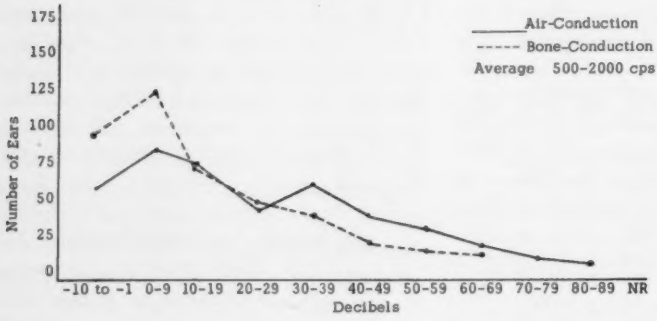

Fig. 3.—Average air-conduction and bone-conduction thresholds for 400 ears.

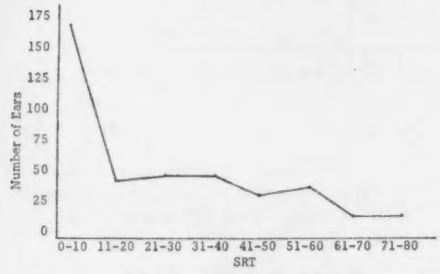

Fig. 4.—SRT scores achieved by 400 ears.

from 20 to 74 years. Seventy-six per cent were between ages 30 and 50 years.

Figure 3 shows the distribution of average air- and bone-conduction thresholds. As revealed by the slope of the curve, the vast majority of the ears could be classed as having only mild or moderate losses of acuity. Two hundred seventy-eight ears, or 70%, fell within 0 and 40 db. loss on air-conduction tests. Eight per cent had losses greater than 40 db., and twenty-two per cent showed air-conduction thresholds of less than 0.

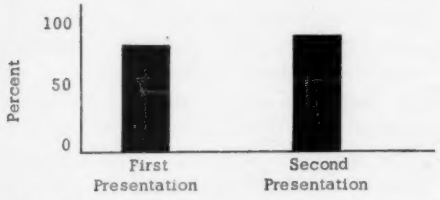

Fig. 5.—Intelligibility scores achieved on first and second presentations.

In line with the air-conduction and bone-conduction findings are the Speech Reception Thresholds shown in Figure 4. Seventy-six per cent of the SRT scores were between 0 and 40 db., with the remaining twenty-four per cent falling between 40 and 80 db.

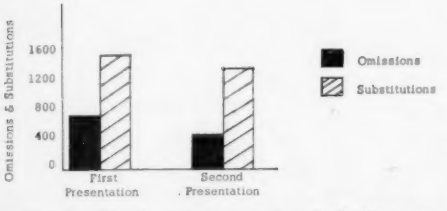

Fig. 6.—Word omissions and substitutions occurring as first and second presentations of PB's were made.

The PB test lists were presented twice to each ear. Figure 5 presents results which indicate that there is a slight gain in the scores achieved on second presentation. On the first presentation, the average score was 89.2%, whereas, on the second, it was 92% for the 400 ears.

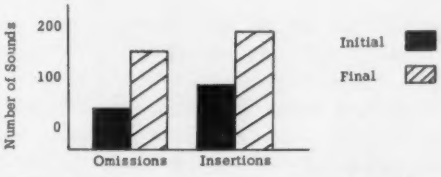

Fig. 7.—Sound omissions and sound insertions according to position for 400 ears on PB words.

Figure 6 presents a comparison of the number of word omissions and substitutions that occurred on first and second presentations. Considerably more substitutions than omissions occurred on both presentations. There was, however, 36.1% decrease in the number of word omissions on the second presentation, as compared with a 21.4% decrease in word substitutions.

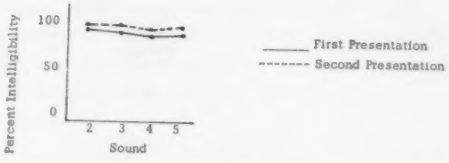

Fig. 8.—Relative intelligibility of 400 ears on PB words on first and second presentations in relation to the number of sounds in the words.

Omissions and insertions which occurred are shown in Figure 7, according to their positions in the words. Approximately seven times as many omissions of sounds occurred in the final as in the initial position, and approximately twice as many sound insertions occurred in the final as in the initial position.

Upon observation of Figure 8, it appears that the number of sounds in the words

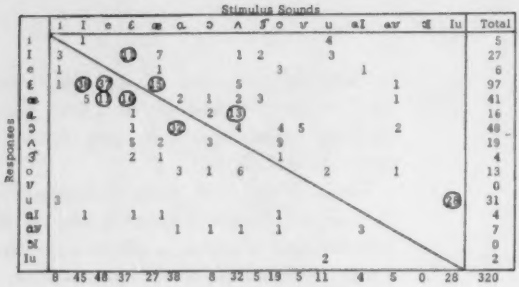

Chart 1.—Vowel confusion occurring in the first presentation of the PB words.

Most Frequent Confusions

Stimulus Sounds	Responses
ɪ	ɛ - 38
e	ɛ - 37
ɑ	ɔ - 32
ɪu	u - 28
ɛ	æ - 16
æ	ɛ - 15
ʌ	ɛ - 13
ɡ	ɪ - 11
o	æ - 11

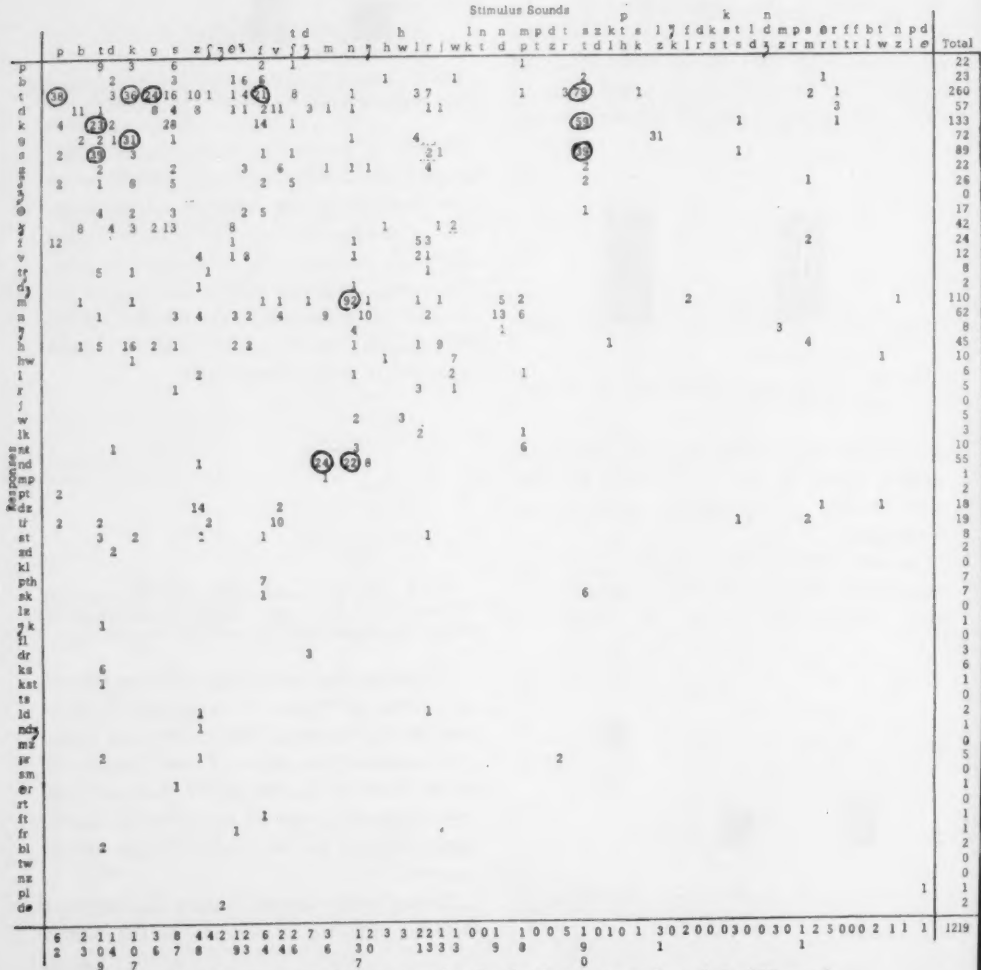

Chart 2.—Consonant confusion occurring in the first presentation of the PB words.

Age	Mean Scores: Conductive Number*	Air Conduction	Bone Conduction	SRT	PB-1	PB-2	Non-Conductive Number*	Air Conduction	Bone Conduction	SRT	PB-1	PB-2
Below 30	4	22.0	3.5	22.0	92.5	94.5	11	15.2	12.9	11.5	89.6	91.8
30-39	33	30.9	38.8	31.8	93.7	95.5	10	18.9	16.8	18.5	84.6	90.8
40-49	14	31.7	3.8	32.4	94.3	96.4	25	40.3	30.1	37.6	82.4	88.3
50-above	4	28.0	2.0	29.5	96.0	96.5	9	27.9	24.9	28.0	86.2	90.7
Average	xx	27.8	11.0	28.9	94.1	95.7	xx	25.6	21.2	23.9	85.7	90.4

*Number totals 55 ears with conductive and 55 ears with non-conductive losses.

Chart 3.—Age ranges and audiometric measures on 55 ears with conductive loss and 55 ears with nonconductive loss. Air-conduction and bone-conduction averages are for 500, 1,000, and 2,000 cps.

of the test lists made little difference in their intelligibility in both first and second presentations. In both instances, however, four-sound words were slightly less intelligible.

Chart 1 presents the vowel confusions which resulted. They were very few in number, totaling only 320 in 20,000 responses. They were greatly scattered, however, as is shown in the rank order. When the vowel (ɪ) is given in the stimulus, (ɛ) is most frequently confused, and likewise, when (e) is in the stimulus word, (ɛ) is most frequently confused. When the vowel (a) is in the stimulus, the (ɔ) vowel is most often confused. When the diphthong (ɪu) is in the stimulus, the vowel (u) is most frequently confused, etc.

The consonant confusions, as presented in Chart 2, are more numerous than the vowels, totaling 1,219. Outstanding confusions were (t) for (p, k, g, f, st), (k) for (t, st), (g) for (k), (s) for (t, st), (m) for (n), and (nd) for (m, n). However, when the percentages of occurrence of error on vowels and consonants are compared, one finds that there is greater percentage of error on vowels (0.016%) than on consonants (0.009%). When a Standard Error of Difference in proportions is computed, by utilization of the following formula,

$$\sigma D = \sqrt{\frac{pq}{N_1} \pm \frac{pq}{N_2}}$$

it is found that a significantly greater proportion of errors occurred among vowels than consonants (C. R., 8.88).

VI. Part II. Results and Analysis

The purpose of taking a closer look at results from the 110 ears (55 conductive losses and 55 nonconductive losses) was to attempt to determine whether the types of errors associated with either classification of hearing loss differed greatly from the other.

Stimulus	Non-Conductive: Confusions	Number	Percent	Conductive: Confusions	Number	Percent
I	e-16, u-1, æ-1	18	21.4	ɛ-4	4	8.5
e	aI-1, æ-4	5	5.6	ɛ-3	3	6.4
ɛ	I-2	2	2.4	æ-4, I-2	6	12.8
æ	e-2, ɛ-3, ɔ-2	7	8.3	I-4, ɛ-6	10	21.3
a	ɔ-30, aʊ-1	31	36.9	ɔ-9, o-5	14	29.8
ʌ	e-5, a-2, ɔ-4	11	13.1	----------		
ɔ	ʊ-1, aʊ-1	2	2.4	----------		
o	ʌ-2, ɔ-3, aʊ-1	6	7.1	----------		
ʊ	----------			a-7, o-3	10	21.3
Iu	u-2	2	2.4	----------		
	Total	84		Total	47	

Chart 4.—Comparison of vowel confusions.

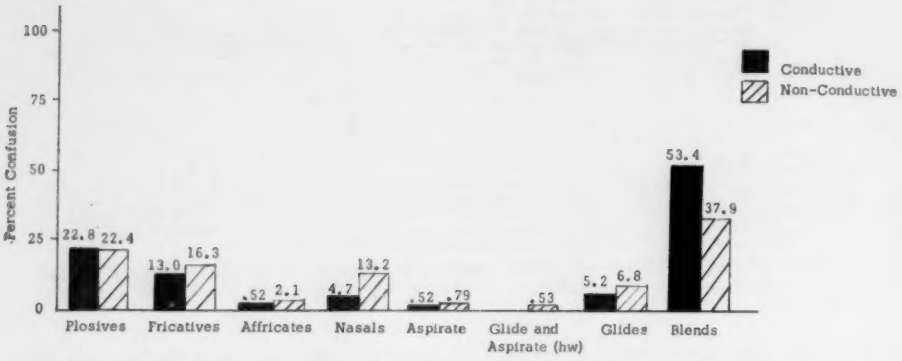

Fig. 9.—Percentage comparisons of consonant confusions for those with conductive and nonconductive losses relative to specific sound groups.

Chart 3 shows averages of air-conduction, bone-conduction, SRT, and PB scores for both categories of loss, by age. Of interest to note is the lower SRT average for the nonconductive group (23.9 for nonconductive and 28.9 for conductive), as compared with the difference in PB averages.

Chart 4 shows comparison of the vowel confusions which occurred relative to each hearing-loss category on first presentation of the PB words. One observes that there are almost twice as many confusions occurring within the nonconductive classification as in the conductive (total words in each category are 2,750). Greatest confusion for both categories occurred on vowel (a). It is interesting to observe that no confusions occurred on the (u) vowel for the nonconductives, whereas almost one-fourth of the total confusions were made on this vowel by the conductives. Almost one-fourth of the errors made by the nonconductives occurred on (ɪ), as compared with 8.5% by the conductives.

A percentage comparison of consonant confusions is presented in Figure 9. It was found that the total number of confusions made by the nonconductive group was 380 and by the conductive group only 193. Greatest percentage of error for both groups occurred on the (st) blend. Casual inspection reveals that the order of magnitude of confusion is not vastly different for any group of sounds except for the blends, with the conductives showing a substantially greater percentage of confusion (53.4%, as compared with 37.9% for the nonconductive group), and the nasals, with the nonconductives showing almost three times

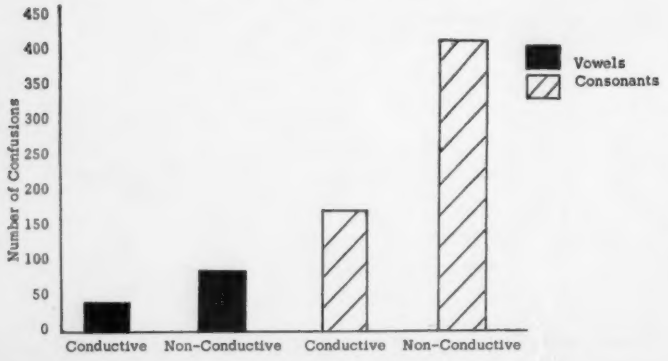

Fig. 10.—Comparison of vowel and consonant confusions occurring on presentations of PB words in relation to types of hearing loss (5,500 responses per hearing-loss category).

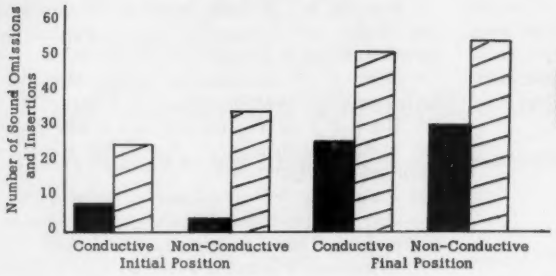

Fig. 11.—Sound omissions and insertions occurring in initial and final positions on presentations of PB words to the 55 ears with conductive losses and to the 55 ears with nonconductive losses (5,500 responses per hearing-loss category).

the percentage of confusion the conductives show.

Figure 10 presents a summary picture of the vowel-consonant confusions which occurred, in relation to hearing-loss categories. Vowel confusions are considerably fewer than consonant confusions in number. The nonconductives show more confusions for vowels and consonants than do the conductives. A Standard Error of Difference in proportion computed between vowel and consonant errors reveals that there is a significantly greater proportion of errors among vowels than consonants (C. R., 12.25).

Figure 11 shows sound omissions and insertions which also demand attention in a structural analysis of errors in a discrimination task. The reader will note that insertions are more frequent than are omissions and, regardless of hearing loss classification, the final sounds in a word are generally more vulnerable to these types of errors than are the initial sounds.

Upon close inspection of the data, the following questions arose: How frequently do subjects of the two hearing loss categories repeat the same error response when the PB words are presented a second time? Is there noticeable variation in error repetition among age groups?

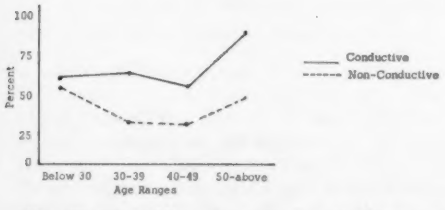

Fig. 12.—Repetition of errors on second presentation of PB words.

According to the plot in Figure 12, those with conductive losses show that through age groups they will repeat the first error response more frequently when presented with the PB words a second time than will those with nonconductive losses. The nonconductives tend to omit, substitute a different error response, or make a correct response. In other words, those with nonconductive type hearing losses are not as consistent in their errors as are those with conductive type loss.

VII. General Conclusions

In answer to the questions set forth at the outset, the following observations pertain for the data analyzed:

1. Sound substitutions occur more frequently in the discrimination task than do omissions and insertions.

2. Confusions within groups of consonants are very similar for both hearing-loss categories except for nasals and blends. It appears that somewhat less confusion occurs on blends by the nonconductive group whereas somewhat more confusion occurs on the nasals.

3. The PB words containing two, three, four, and five sounds showed no marked differences in intelligibility.

4. Sound omissions and insertions in error responses occur most frequently in the final position of the word.

5. Discrimination losses decrease when the task is presented a second time.

6. Although vowel confusions occurred less frequently than did consonant confusions, a significantly greater proportion of confusions occurred among vowels than among consonants.

7. There is a difference in the degree to which the subjects with conductive loss and those with nonconductive losses will persevere in their error responses. Those with conductive loss were more consistent at each age level in repeating their errors than those with nonconductive loss.

Speech and Hearing Clinic, The Ohio State University (10).

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Bone-Conduction Thresholds in Stapes Surgery

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The measurement of pure-tone bone-conduction thresholds figures prominently at present in the selection of cases for stapes surgery for otosclerotic deafness and in the prediction of postoperative results.[1]

In the period preceding the development of modern stapes surgery, the importance of the mechanical shift in bone-conduction thresholds following successful fenestration surgery of the horizontal canal was accepted generally along the lines suggested by Carhart, Shambaugh, and McConnell.[2,3] The average improvement in these thresholds as postulated by Carhart was as follows: no shift at 250 cps, 5 db. at 500 cps, 10 db. at 1,000 cps, 15 db. at 2,000 cps, and 5 db. at 4,000 cps. A carefully controlled study by McConnell and Carhart yielded somewhat different values of shifts following successful fenestration nov-ovalis, namely: 1.2 db. at 250 cps, 1.4 db. at 500 cps, 6.5 db. at 1,000 cps, 7.4 db. at 1,500 cps, 8.5 db. at 2,000 cps, 6.4 db. at 3,000 cps, and 4.2 db. at 4,000 cps. When statistical tests of significance were applied by them to these data, only the shifts at 1,000 cps through 4,000 cps were found to be attributable to surgery. An interesting point made by the foregoing authors was that there was slightly more shift in "B" and "C" cases than in "A" cases. That is, the greater the initial threshold loss for bone conduction, the greater the improvement in bone-conduction threshold following successful surgery.

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In fenestration surgery of the horizontal canal, the bone-shift values suggested by Carhart were applied as a correction to the bone-conduction thresholds of patients being considered for the surgery. According to McConnell and Carhart, the minimal conductive deficit believed to be due to the disruption of the ossicular chain during fenestration surgery is 20 db. at all frequencies from 125 through 4,000 cps and 15 db. at 8,000 cps. These figures were added to the corrected bone-conduction thresholds to yield a predicted postoperative air threshold.

With the development of present-day stapes mobilization, it became apparent that the minimal conductive deficit need no longer be included in the prediction of postoperative thresholds, since it now was theoretically possible to restore the normal action of the middle ear mechanism, thereby eliminating the air-bone gap. The question of whether there would be a shift of bone conduction thresholds, however, continued to interest surgeons to the extent that such shifts could be related to postoperative air-conduction thresholds.

The first statistical report on stapes mobilization published by us showed that in "A" cases, or those with the best preoperative bone thresholds, successful mobilization was followed on the average by small *decreases* in bone conduction sensitivity.[4] It was suggested that these apparent decreases in bone thresholds may have been due to the normal and near-normal hearing by air conduction which resulted from the surgery and the consequent masking of the test frequencies by noise in the test room. In the "B" and "C" cases some improvement in bone thresholds following successful mobilization was noted.

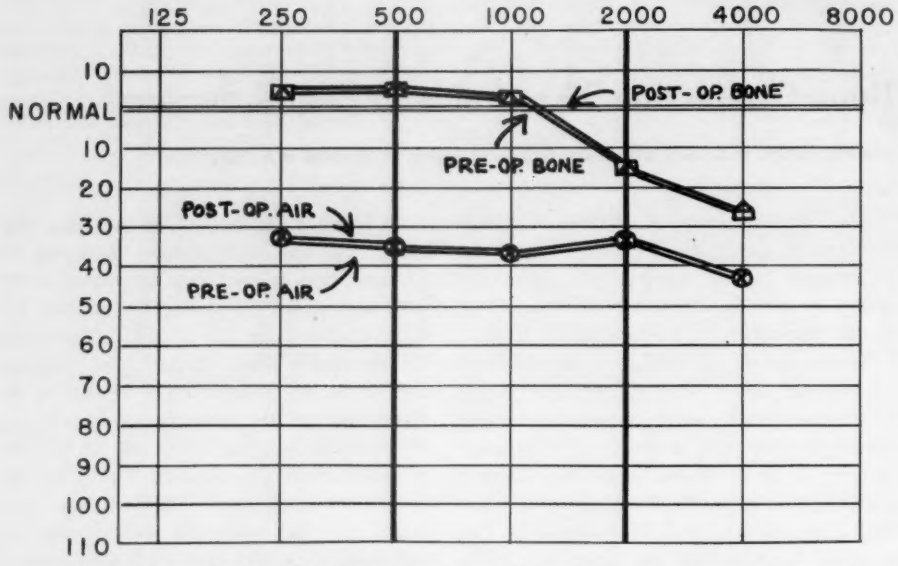

Fig. 1.—Bone-conduction shifts in improved cases. Average pre- and postoperative air- and bone-conduction audiograms of 36 non-operated-on ears (Table 2).

The present study was undertaken to examine this matter closely with careful control of equipment and test personnel, to determine whether any systematic correction should be applied to the preoperative bone-conduction thresholds in selecting cases for stapes surgery and in predicting maximum surgical results.

The cases noted as improved in this study are those in which there was an improve-

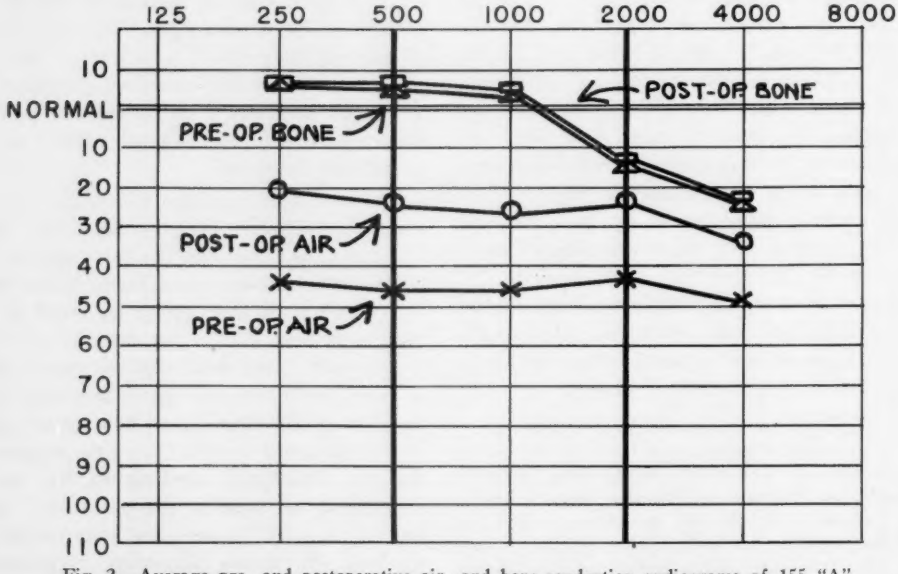

Fig. 2.—Average pre- and postoperative air- and bone-conduction audiograms of 155 "A" and "B" cases (Table 3).

TABLE 1.—*Comparison of Bone Thresholds with Air Thresholds* *

	Frequency				
	250	500	1,000	2,000	4,000
Mean variation of bone from air threshold, db.	2.25	0	4.5	—2.25	0.87
Standard deviation	5.15	4.9	5.36	4.7	5.02

* Forty ears with sensorineural deafness were tested on the audiometer used in this study. Negative figures indicate bone thresholds worse than air thresholds. All others indicate bone better than air.

TABLE 2.—*Variations in Bone Thresholds in Thirty-Six Non-Operated-On Ears* *

	Frequency				
	250	500	1,000	2,000	4,000
Mean variation pre- to postoperative, db.	0.8	0.0	—0.1	—0.6	—0.7
Standard deviation	5.01	4.93	4.90	4.87	4.86

* Before and after successful stapes surgery was performed on the opposite ears.

ment in the air-conduction threshold averaging more than 10 db. at the frequencies 500, 1,000, and 2,000 cps. "A" cases are those in which the preoperative bone conduction thresholds were no worse than 10 db. at any of the frequencies 500, 1,000, and 2,000 cps. "B" cases are those in which the preoperative bone-conduction thresholds did not meet the criteria for "A" but were no worse than 20 db. at 500 or 1,000 cps or 30 db. at 2,000 cps. Bone-conduction threshold losses greater than the foregoing levels placed a case in category "C."

All test data were obtained not less than one month nor more than five months after surgery.

Equipment and Calibration

All tests, both pre- and postoperative, were conducted on an Audivox 7B audiometer, which was checked periodically for electrical and acoustical performance with an Allison 3-A Audiometer Calibration Unit. Both output-level and attenuator-linearity checks were made to establish the stability of the audiometer up to the bone-conduction oscillator. The calibration and stability of the bone unit itself were determined by (1) testing patients who had "pure" sensorineural deafness and calculating the agreement between their air and their bone thresholds at each frequency (Table 1) and (2) by calculating the bone threshold shifts for the un-operated-on ears of the subjects whose operated-on ears had been improved by the surgery (Table 2).

The standard deviations of approximately 5 db. shown in Table 1 are within general clinical experience. An analysis of office data collected at the beginning of the period under study, January, 1956, and at the end, April, 1958, indicated similar data.

As a check on the stability of the bone-conduction thresholds of the subjects of this study, the variation in the bone thresholds of the un-operated-on ears in 36 of the improved cases was determined as shown in Table 2.

TABLE 3.—*Gains in Bone Conduction Sensitivity in Cases of Improved Hearing Following Stapes Surgery* *

	Frequency					
	250	500	1,000	2,000	4,000	Cases, No.
"A" cases						55
Average gain, db.	—0.82	—0.73	0.18	—0.73	—0.55	
Standard deviation	4.87	4.88	4.97	4.88	4.90	
"B" cases						100
Average gain, db.	2.0	0.7	1.8	3.7	3.5	
Standard deviation	5.17	5.04	5.15	5.34	5.31	
"A" & "B" cases combined						155
Average gain, db.	1.0	0.2	1.3	2.1	2.1	
Standard deviation	5.08	5.0	5.1	5.2	5.2	

* Negative signs indicate loss of sensitivity.

In view of the results arrived at earlier (Rosen and Bergman [1]) in which the postoperative improvements in bone thresholds for the "B" cases were greater than for the "A" cases, the data were again divided into these categories. Fifty-five improved "A" cases and one hundred improved "B" cases were included in this study. The changes in bone-conduction thresholds from the pre-to the postoperative audiograms are shown in Table 3 and in Figures 2, 3, and 4.

One method of studying the validity of the bone shifts obtained on the operated-on ears is to compare these shifts with those recorded on the non-operated-on ears. This comparison is seen in Table 4.

Since it could be thought that a single tester might influence the results of the

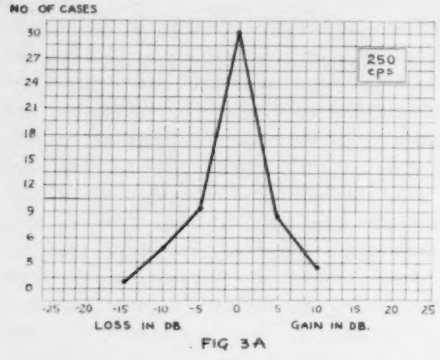

FIG. 3A

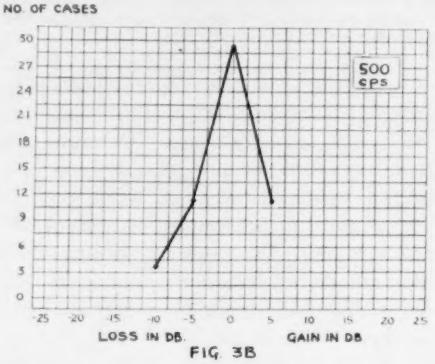

FIG. 3B

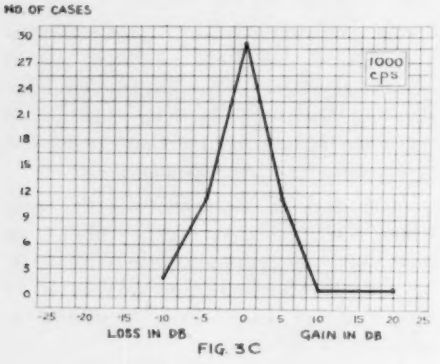

FIG. 3C

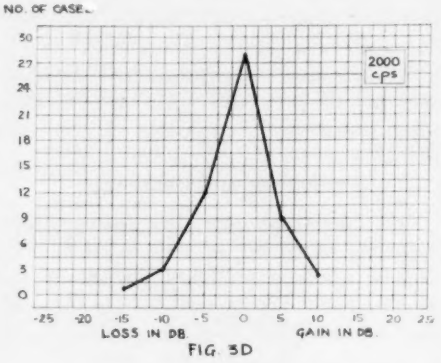

FIG. 3D

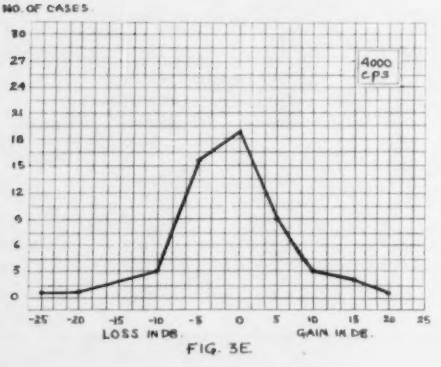

FIG. 3E.

Figs. 3A-3E.—Distribution of shifts in bone-conduction thresholds at each frequency in 55 "A" cases, with improved hearing following stapes surgery.

TABLE 4.—*Comparison of Changes in Bone Thresholds on Successfully Operated-On Ears.*

	Frequency									
	250		500		1,000		2,000		4,000	
	M	SD	M	SD	M	SD	M	SD	M	SD
Op. ears	1.0	5.08	0.2	5.0	1.3	5.1	2.1	5.19	2.1	5.19
Non-op. ears	0.8	5.01	0.0	4.93	—0.1	4.9	—0.6	4.87	—0.7	4.86
Difference	0.2	0.07	0.2	0.07	1.4	0.2	2.7	0.32	2.8	0.33

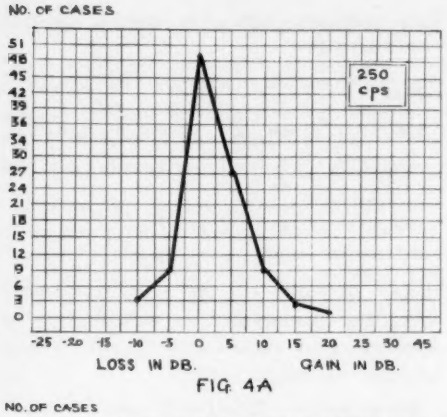

FIG. 4A

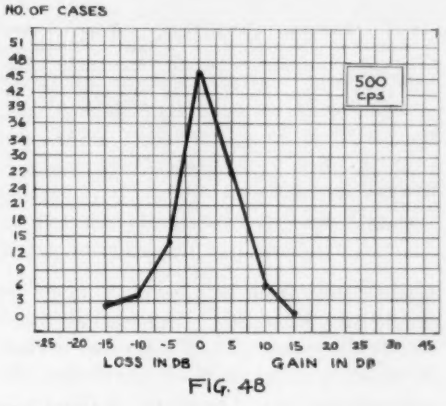

FIG. 4B

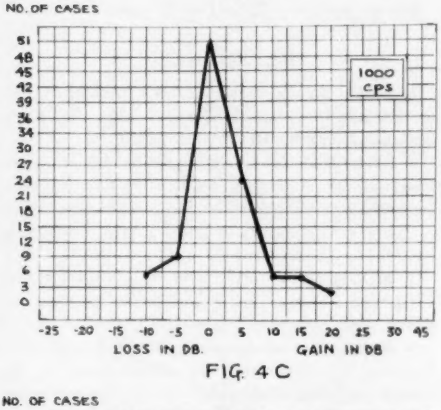

FIG. 4C

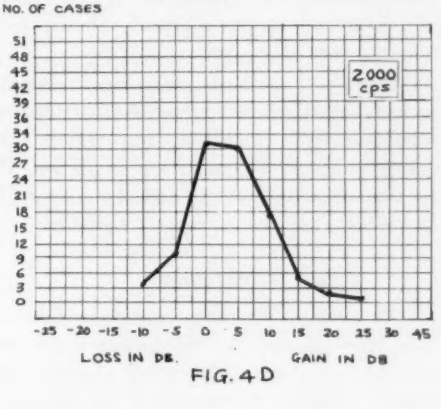

FIG. 4D

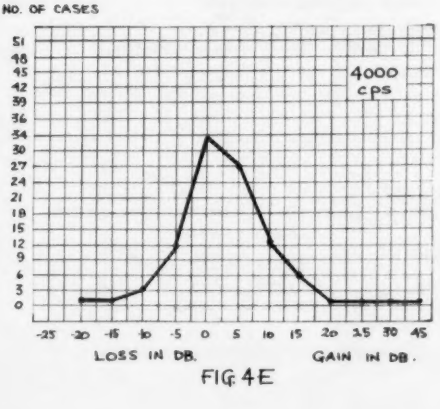

FIG. 4E

Figs. 4A-4E.—Distribution of shifts in bone-conduction thresholds at each frequency in 100 "B" cases, with improved hearing following stapes surgery.

TABLE 5.—*Comparison of Bone Threshold Changes Obtained by One Tester with Those Obtained by Two Testers*

	Frequency					
	250	500	1,000	2,000	4,000	Cases, No.
1 tester	1.0	0.2	1.3	2.1	2.1	155
2 testers	−0.9	−0.2	−1.7	1.6	2.1	37

TABLE 6.—*Comparison of Bone-Threshold Shifts in Stapes Surgery Cases (Rosen) and in Fenestration Nov-Ovalis Cases*[4]

	Frequency					
	250	500	1,000	2,000	4,000	Cases, No.
Stapes cases	1.0	0.2	1.3	2.1	2.1	155
Fenestrated nov-ovalis cases	1.2	1.4	6.5	8.5	4.2	58
Difference	0.2	1.2	5.2	6.4	2.1	

postoperative bone thresholds in favor of agreement with his preoperative bone thresholds, a series of 37 cases was studied in which a different tester performed the postoperative test. The results in this series are compared to those already shown for one tester (Table 5).

Finally, since there have been several articles in which the Carhart formulation is advocated for predicting the results of stapes surgery, a comparison is shown of the data obtained by us and that obtained by McConnell and Carhart (Table 6).

Conclusion

The changes in bone-conduction thresholds from the pre- to postoperative tests in 155 cases of improved hearing following stapes surgery are too small to influence meaningfully the selection of cases or to constitute a "correction factor" in the prediction of results in this type of surgery. The data obtained here do not support the application of the "Carhart shift" values to cases of stapes surgery.

101 E. 73d St. (21) (Dr. Bergman).

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Case Reports

Relief of Vertigo by Stapes Mobilization in a Patient with Otosclerosis

HOWARD D. COHEN, M.D., Wilmington, Del.

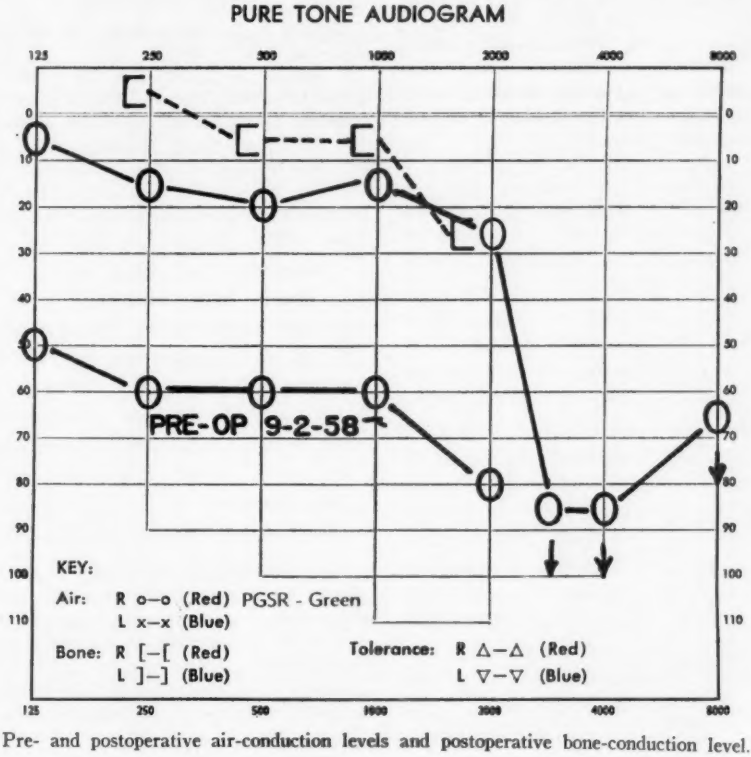
Review of the recent literature reveals that there have been no cases reported in which vertigo accompanied by otosclerosis has been successfully treated by stapes mobilization, although Jordan presented one such case in his course on Ménière's disease at the 1957 meeting of the American Academy of Ophthalmology and Otolaryngology. Jack mentioned relief of vertigo by stapedectomy, but he did not state that it was accompanied by otosclerosis.

Submitted for publication March 10, 1959.

Resident in Otolaryngology, Temple University Medical Center. From the Department of Otorhinology, David Myers, M.D., Professor and Head of the Department; The Vertigo Clinic, Martin Spector, M.D., Director, and The Audiology Section, Philip E. Rosenberg, Ph.D., Director.

Report of Case

A 57-year-old white man was admitted to the Temple University Medical Center on Sept. 23, 1958, with the chief complaint of severe dizziness.



Pre- and postoperative air-conduction levels and postoperative bone-conduction level.

His first attack had occurred approximately 10 years prior to admission, and these attacks had recurred at yearly intervals until July 3, 1958, when he began to experience severe bouts of vertigo lasting three to seven days. His previous history revealed a known hearing loss in his right ear of 25 years' duration, with no recent progression. He had had occasional mild tinnitis in the right ear, but there was no history of otalgia or otorrhea in either ear.

Physical examination revealed normal tympanic membranes. The Eustachian orifices appeared to be normal on nasopharyngoscopy. The Weber test showed lateralization to the right at 256, 512, and 1,024 cps, and the Rinné test was negative in the right ear for the above frequencies.

Audiometry revealed an average loss by air for the three speech frequencies of 67 db. in the right ear and 27 db. in the left ear. Average bone-conduction loss for the speech frequencies was 32 db. in A. D. and 15 db. in A. S. There was an incidental finding of a marked loss in both ears at 3,000 and 4,000 cps due to acoustic trauma. Speech reception thresholds corroborated the pure-tone thresholds, and the discrimination scores were good.

There was transitory first-degree nystagmus on right and left lateral gaze, which was absent on two other occasions and present, but fatigable, on a fourth observation. There was no positional nystagmus at the time of examination, but a history of postural vertigo had been elicited on stooping, arising in the morning, turning to the left, straightening, and looking up.

Minimal caloric stimulation (10 cc. water at 80 F) produced a slightly prolonged reaction in the right ear and a normal response in the left ear.

On Sept. 26, 1958, a right stapes mobilization was performed by me, and otosclerosis, Grade III (Bellucci), was found. Otosclerotic foci were found, extending from the inferior edge of the anterior crus to the promentory and along the anteroinferior border of the footplate, extending across the stapedovestibular articulation. The footplate was fenestrated anterosuperiorly. The foci were separated with a chisel, and the stapes became mobile. Hearing improved immediately.

The postoperative course of the patient was uneventful, and he has had no recurrences of vertigo up to the time of writing. Hearing in the right ear, averaged for the three speech frequencies, is improved 47 db., from 67 db. to 20 db., by air. Bone-conduction levels were improved 20 db., from 32 db. to 12 db., more than was predicted by the Shambaugh-Carhart formula.

Summary

A case of vertigo in a patient with otosclerosis, which was relieved by stapes mobilization, is described.

1003 Delaware Ave. (6).

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Progress in Otolaryngology

Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Internal Ear

Summaries of the Bibliographic Material Available for 1955

BRUCE PROCTOR, M.D., Detroit
With the Collaboration of Michel Portmann, M.D., Bordeaux, France; Enrico Bozzi, M.D., Milan, Italy; Jerzy Szpunar, M.D., Krakow, Poland, and Thea A. Hennecke, M.D., Windsor, Ont., Canada

(*Continued from page 270*)

Vertigo

Kos states that, in the diagnosis of vertigo, an auditory survey is most important. In addition, there should be an otoscopic and ophthalmic examination, x-rays of the internal auditory meatus, and caloric and neurologic examinations. It is important to differentiate between peripheral and central types of dizziness. Nearly all peripheral vestibular disturbances are accompanied by hearing loss; less frequently are the central ones. Recruitment, the Bekesy type audiogram, and auditory threshold fatigue are all helpful in differentiating between cochlear and neural lesions. In cochlear lesions there is recruitment, a short Bekesy excursion, and absence of threshold fatigue. Labyrinthine ischemia gives a high-frequency inner ear type of hearing loss which does not fluctuate and may or may not show recruitment. It is usually bilateral. Hearing loss is of high frequency and never fluctuates but progressively deteriorates. It is commonly associated with arteriosclerosis. In labyrinthian apoplexy, the vertigo is severe and constant and gradually decreases over a period of months as the other labyrinth takes over. There is severe unilateral hearing loss. Hearing loss is the most frequent initial symptom of acoustic neuroma. There may be dizziness but not recruitment, and the threshold fatigue is notably present. Multiple sclerosis shows slight hearing loss, though vertigo may be severe. There is not recruitment but excessive threshold fatigue. These examples illustrate the importance of hearing-loss evaluation in the determination of the cause of vertigo.

Disturbance of position in space requires disease of the body systems of the labyrinth, the ocular or the proprioceptive, writes Hoople. Balance is impossible if two of the three are not functioning. Thus, closing the eyes, as in the Romberg test, will cause loss of balance if proprioception or labyrinth is diseased. True turning, rather than staggering, always indicates a disease of the labyrinth or its intracranial connections. Examination must include complete audiometric and calorigram study of labyrinth function. Spontaneous and persistent nystagmus is usually central, since peripheral labyrinthian nystagmus is fatigable. Explosive and sudden dizziness is usually peripheral. Intracranial dizziness is gradual and increasing, with no periodicity. Low-sodium regimen relieves cases due to edema. Dimenhydrinate, U. S. P. (Dramamine) is effective for symptoms if given parenterally.

Submitted for publication Nov. 10, 1958.

Associate Professor of Otolaryngology, Wayne State University (Dr. Proctor); Professor Agrégé à la Faculté de Médecine, University of Bordeaux (Dr. Portmann); Prof. Dott, University of Milan (Dr. Bozzi), and Medical Academy Docent in Otolaryngology, University of Krakow (Dr. Szpunar).

Marconi discusses a treatment of vertigo with Chinine Sulfite (0.003 gm. per day), epinephrine (30 to 60 drops per day), atropine, vitamins PP and A in big doses, antihistamine, magnesium sulfate, etc. During the main vertigo crisis, the patient should remain in a quiet and dark room, while 0.5 mg. of atropine and 10 cc. of a solution of magnesium sulfate, 15%, or glucosed hypertonic serum is administered intravenously. Should these medicaments not be available, a light jet of cold water into the ear where the nystagmus happens to be might be sufficient to stop the crisis.

Diagnosis and therapy of vestibular neuronitis are discussed by Pfaltz. This condition, first defined by Dix and Hallpike, in 1952, is characterized by dizziness, abnormal caloric irritability of the labyrinths, and diminished galvanic excitability. It is differentiated from Ménière's disease by the absence of auditory and neurologic disturbances. Apparently the condition consists of inflammatory changes in the peripheral vestibular neuron on the basis of foci in the sinuses, tonsils, and ears or generalized infections. The author examined 25 patients with a history of dizziness, nausea, headache, and disturbances of equilibrium. No auditory disturbances were present. Underlying conditions were maxillary sinusitis (13 cases), influenza, chronic nephritis, chronic salpingitis, hepatitis and gastrointestinal infections (two cases each), and recurrent tonsillitis and epidemic parotitis (one case each). The following objective results were found: normal hearing in 14 cases, defects in higher frequencies not related to the vestibular disturbance in 11, bilateral caloric hypoexcitability in 2, unilateral hypoexcitability in 21, and a tendency toward nystagmus in 2. No spontaneous nystagmus was found; there was no hypertension. In 17 patients the symptoms disappeared after healing of the underlying condition; in 9 the caloric excitability returned to normal. The history and otoneurologic findings in this series were almost identical to those encountered by Dix and Hallpike. These cases, therefore, represent a well-defined syndrome, a toxic or inflammatory lesion of the peripheral vestibular neuron similar to the well-known retrobulbar neuritis, which has been observed in connection with acute infections and intoxications and which also heals after the underlying disease has disappeared. It can be assumed that vestibular neuronitis is an inflammatory-toxic-allergic condition in which there is either direct damage to the peripheral neuron due to bacterial toxins or an indirect inflammatory reaction within the neuron on the basis of an allergy to endotoxins. Therapy consists of elimination of the underlying disease, application of antihistamines, decrease of vascular permeability with vitamins P and C, restriction of fluid intake and of Na ions, and administration of drugs influencing peripheral circulation.

A number of cases are described by Behrman, showing a recurrent transient postepileptic vertiginous state, and arguments are advanced suggesting that this syndrome is a variant of vestibular epilepsy, i. e., a form of epilepsy in which seizures are provoked by excessive, spontaneously arising discharge of the vestibular apparatus. Evidence is also furnished, indicating that vestibular epilepsy is a subgroup of sensorily precipitated epilepsy, i. e., epilepsy provoked under certain circumstances in constitutionally predisposed persons by the stimulation of various types of receptors. The clinical features, differential diagnosis, and therapy are discussed.

Beretta and Livan report the results obtained by a treatment of polyvinylpyrrolidone in 10 cases of serous labyrinthitis in the course of acute purulent and chronic otomastoiditis, in 4 cases of postoperative serous labyrinthitis, and in 9 cases of labyrinthine fenestration. In all patients treated with polyvinylpyrrolidone the therapy caused the disappearance of labyrinthine symptomatology in a short time, together with the shortening of the clinical course. The results obtained by the polyvinylpyrrolidone treatment prove the efficient action of

this drug. The quick exchange caused by the drug in the labyrinthine liquids, besides a decompressive action, tends to the rapid elimination of toxic products and to a better nutritious support of the neuroepithelium immersed in such fluids. Furthermore, the nontoxic action appearing at the level of the inflammation focus contributes to the improvement of the clinical course noted in the treated cases. During a long experience in the use of polyvinylpyrrolidone, the authors never noticed any disturbance due to it. The authors suggest that the drug should become very commonly used. Obviously the treatment is to be considered mostly symptomatic, as it will only flank and not replace removal of the cause when known.

In 21 normal subjects, Bartalena studied the persistence of the nystagmus induced by rotatory stimulation (Bárány's test) and by caloric stimulation (Veit's test) after the administration of antihistaminic drugs with an increasing daily dosage. From a general point of view it seems that the antihistaminic drugs decrease the vestibular reaction—the higher the antihistaminic drug dosage the lower the vestibular reaction. In the discrimination of the cases it appears that the vestibular reaction is frequently in correlation to the antihistaminic drug dosage or the method of stimulation employed (rotatory or caloric test). In conclusion the author states that the effect of the diphenhydramine on the vestibular apparatus is in correlation with several peripheral and central reactions that modify the function of the neurovegetative system.

After summarizing present knowledge about the pharmacologic action and use of Cimicifuga racemosa in the treatment of vertigo and auricular humming syndromes, Salerno reports the results of experimental enquiries according to which he could demonstrate that (1) C. racemosa has neither peripheral nor central action on labyrinthine reflexes and (2) that this drug, owing to the active nonhydrosoluble resinous principles contained therein, has a general hypotensive action, remarkable for its intensity and duration. Therefore, C. racemosa would find its elective use in vertigo and humming syndromes connected with general or local hypertension states.

Vestibular Tests

Hallpike gives the rationale and technique of the caloric test. The test results are discussed under the following headings: (*a*) interpretation, (*b*) statistical analysis of results obtained in normal subjects, (*c*) results obtained in organic disorders of the vestibular system: (1) Ménière's disease, (2) vestibular neuronitis, (3) tumors of the eighth nerve, and (4) lesions of the cerebral hemispheres.

Warm and cold stimulation of the cupula were used by Aschan to study vestibular function. Nystagmus was recorded and analyzed as to duration, frequency, and amplitude. Cold effects were more prolonged than warmth effects. This is explained on a physical basis, as temperature change produces a change of specific gravity and of viscosity, both affecting mobility and hence flow of endolymph. Ampullopetal and ampullofugal stimuli were found to have the same effect on cupular deviation. This is ascribed to a central coordinating mechanism.

Giulio investigated, in the awake animal, the labyrinthine thermic threshold of the isolated contraction of the rectus externus muscle and of the eye movement in toto in relation to the spatial orientation of the lateral semicircular canal. The following positions were studied: horizontal, inclined at 30, 65, and 90 degrees over the horizontal plane, with the ampulla over. 1. The value of the threshold for the minimal phasic activity of the eye muscle by a punctiform thermic stimulus depends on the orientation of the canal in relation to the horizontal plane. 2. The interrelation is not as strict as it would be by the physical postulates on the origin of the endolymphatic convection currents; however, a null position does not exist. (*a*) In fact, when the canal is horizontally oriented, the threshold appears

to be rather elevated, though not very much elevated. This depends on the secondary curvatures of the bone canal, described by the anatomists. (*b*) The threshold in the canal inclined at 30 degrees over the horizontal line is very low. (*c*) The threshold in the canal inclined at 30 degrees over the horizontal line is, on the contrary, higher than the foregoing one. This depends very likely on the interference on the otolithic ocular reflexes. 3. In the various orientations of the lateral canal, the slow contraction velocity of the rectus externus muscle is a linear function of the logarithm of the temperature increase, measured at the level of the ampullary region of the lateral semicircular canal (that is, of the intensity of the stimulus). Only the "slope" of the straight line changes according to the various orientations of the canal; the slope is minimal when the canal is in the horizontal position; it is much more accentuated when the canal is inclined at 30 or 60 degrees over the horizontal plane (ampulla over). 4. The highest frequency of nystagmus is analyzed. In conclusion, under physiological conditions, a position of the head in which the thermic stimulation is absolutely ineffective does not seem to exist.

According to Giannelli and Pennetta, any eventual variations in the values of the auditory threshold might be ascribed to the action of vestibular stimulation when a correspondence should arise between the width of the vestibular stimulation used (either thermical or rotatory) and the width of the possible audiometric variations. A thermic stimulation was thus applied to two subjects by means of water at 5, 15, and 25 degrees. Three other patients were subject to rotatory stimulation with negative acceleration of 90, 180, and 360 degrees per second. The different tests were tried at different times. The frequencies of 500, 1,000, 4,000, 6,000 cps were considered. These are the results obtained: (*a*) Thermic stimulation: 24 times of 48, determinating variations were remarked in the threshold values (+5/10 db., 18 times; −5/10 db., 6 times). The contemporary nystagmic reply always proved to correspond to the intensity of the applied stimulation; (*b*) Rotatory stimulation: only 10 variations in threshold value were remarked of 36 determinations (+5/10 db., 4 times; −5/10 db., 6 times). The acoustic receptors do not appear, therefore, to be influenced by vestibular stimulation. No connection exists between the width of vestibular modification and the threshold variations.

Unilateral predominance of the provoked vestibular nystagmus is, according to de Reynier, mostly due to a diminution of the homolateral nystagmus, at least in a group of 40 patients with trauma of the head. In 15 cases, the reactions were measured again after 3-36 months. The directional preponderance tends to disappear, and the duration of the nystagmus tends to become normal again. The direction of the preponderance was toward the lesion and from the lesion in about an equal number of cases.

Positioning nystagmus is a nystagmus which follows a quick change of position of the head. It starts after a certain latent time and goes on for a limited period only. Stenger discusses the differences between positional and positioning nystagmus, the influence of position, change in position, and neck reflexes, the central or peripheral origin, etc. The author discusses the problem very thoroughly and finally concludes, on account of experiments on rabbits, that the increase of liquor pressure may provoke paroxysmal phenomena similar to the one described. It is proposed as an explanation for positioning nystagmus.

Miehlke was able to provoke positional nystagmus in a series of rabbits by bringing a minute droplet of blood into the perilymphatic space of the upper part of the labyrinth. In a series of photographs the histological picture is clearly demonstrated. The smallest processes in the perilymphatic space of the superior part of the labyrinth regularly provoked positional nystagmus in experiments on animals. In these experiments, the author corroborated the findings of Versteegh and Jongkees

about destruction of the sacculus. Operations upon the inferior part of the labyrinth do not have any influence on spontaneous movements and position. They do not cause positional nystagmus.

The advantages of a new method of rotational stimulation of the vestibular apparatus are the following, according to Arslan: (1) application of a single stimulus (an angular acceleration) on the vestibular apparatus, this stimulation not being troubled by a preliminary acceleration in the opposite direction, as is the case with the method of Bárány; (2) exact knowledge of the value of the applied stimulus, that is to say, knowledge of the degree of angular acceleration; (3) fixation of the head of the subject in a fixed plane, so as to obtain vestibular reflexes (nystagmus etc.) exclusively from the stimulated semicircular canal. The results obtained by this exact method of stimulation in certain typical cases of vestibular diseases are evaluated.

Bekesy has investigated the subjective sensation of observers seated in a chair that could be rotated around a vertical axis. Not only rotation but also an apparent horizontal displacement was felt, which is explained as a function of the otoliths. Favoring this assumption of two different mechanisms is the fact that the apparent displacements and rotations can adapt or recover independently of each other. Under continuous sinusoidal stimulation the sensation of displacement fades away faster than does the sensation of rotation. Furthermore, the threshold of apparent displacement may differ considerably from the threshold of apparent rotation. Repetition of rotatory stimuli results in a diminution of the sense of rotation not only for very strong but also for physiological stimulations and for those near the threshold. In experiments on the threshold of sensation for rotation stimuli, it was found that, when the amplitude is increased from subliminal values, the observer feels first a pure rotation; only upon further increase of amplitude does he feel a sensation of displacement as well as rotation. Control tests showed that sitting still for 20 minutes in a stationary chair does not result in a rise in the threshold; it is not immobility but oscillations of the chair that raise the threshold. This is especially true of sinusoidal rotary oscillations of the kind that makes babies sleep. Strong, irregular movements on the part of the observer may have the opposite effect on the threshold. The following method was used to measure the difference limen above the threshold: During half of the rotary oscillation of the chair, the amplitude of the oscillation could be increased or decreased by the observer. The measurements of the resonant frequency of the cupula seem to make it possible to determine when a mechanical change of the vestibular organ is involved and when only the nervous system is involved. Thereby mechanical and neural changes may be discriminated.

If a subject is rotated and the rotation suddenly stopped, two phases of after-sensation and nystagmus can be distinguished which go in opposite directions. The nature of these phenomena is investigated by Aschan and Bergstedt by replacing rotatory stimulation by caloric stimulation of the cupula. Results indicate a process in the C. N. S. which may add to or subtract from the peripheral vestibular mechanism and which causes the second phase of the post-rotation syndrome.

An attempt at quantitative evaluation of nystagmus and of the movement of the cupula following various types of cupular stimulation in 30 human subjects is reported by Aschan. It is shown that nystagmus does not quantitatively correspond to turning sensation. With cessation of angular acceleration there is a reduction of speed of the slow phase of nystagmus, which is considered an indication of cupular movement. It is explained as displacement of the cupula with sliding over the surface of the crista and a slower movement back to rest position.

Previously Aschan had shown that humans exposed to angular deceleration show an immediate reduction of the speed of the slow component of the nystagmus. Accord-

ing to Aschan, this was ascribed to torsion of the cupula and a sliding movement of the cupula along the surface of the crista. Now it is shown that the same phenomenon occurs in rabbits and pigeons, the first type of movement being associated with weaker stimulation and the latter with stronger stimulation.

The correlation between the speed of the eye in the slow phase of nystagmus and vestibular stimulus was carried out by Henriksson. The results obtained can be summarized as follows. During constant acceleration, the speed of the eye in the slow phase of vestibular nystagmus varies linearly with the acceleration of the rotation, and with the duration of the stimulus; normally, the angle speed of the eye during the slow phase of nystagmus equals the rotation of the head if the subject is accelerated with his eyes open and uncovered; when the subject is blindfolded during acceleration the vestibular eye speed is usually less than that of the skull; i. e., the vestibular stimulus by itself is not sufficient completely to compensate for the rotation; the vestibular eye speed produced by a given stimulus is practically constant for a given person but varies from one person to another; when the same sort of stimulus as in cupulometry is used, the speed of the eyes is not correlated with the duration of the nystagmus.

Aschan, Bergsted, and Stahle describe a very thorough investigation into nystagmography. They prove that it can form a part of routine neurotological examination owing to the fact that nystagmus, as a spontaneous sign of vestibular disturbance, can be recorded in many cases where it would otherwise be overlooked and that recording makes possible a quantitative assessment of a variety of nystagmus phenomena which are not measurable without registration (e. g., the eye speed). The calibration is of great importance and is amply dealt with. A second advantage of nystagmography by means of electrical potentials is the possibility of avoiding fixation. Even Frenzel's spectacles do not prohibit the fixation completely. It is necessary to examine in the dark. The study has included a survey of nystagmus as a spontaneous sign. This phenomenon appears to be much more frequent than it is thought to be on account of visual inspection. Nystagmus has been examined, too, as a sequel to toxic states. Especially the study about positional nystagmus following alcohol intoxication is very revealing. Also, an examination is made of nystagmus during rotatory tests, especially cupulometry. Cupulometry in cases with unilateral loss of vestibular function produces a postrotatory preponderance away from the diseased ear, as revealed by observation with the aid of Frenzel's spectacles. Nystagmography, with the patient's eyes closed, does not show any preponderance; equal ampullopetal and ampullofugal stimuli result in postrotatory nystagmus of equal duration. Thus no evidence is obtained that Ewald's law is applicable to man. Finally, nystagmography is performed during calorization in normal persons and patients. If spontaneous nystagmus is already present before irrigation is commenced, quantitative assessment of the caloric reaction is facilitated by taking the spontaneous nystagmus record as baseline.

Nystagmus can best be classified as vestibular and nonvestibular (ocular), write Aschan and Bergstedt. The latter includes ocular movements stimulated from the eye, the optic nerve, the ocular muscles, or their central connections. Nonvestibular nystagmus may be divided into fixation and gaze nystagmus. The term "latent nystagmus" should be reserved for monocular fixation nystagmus. Nystagmography may differentiate the types of nystagmus from each other, since it shows objectively that (1) fixation nystagmus often disappears completely in the absence of fixation, while vestibular nystagmus becomes more pronounced; (2) fixation nystagmus is not summated on induced vestibular nystagmus; instead one obtains two patterns superimposed on each other (spontaneous and induced vestibular nystagmus, on the other

hand, become summated to form a uniform tracing); (3) spontaneous vestibular nystagmus can be revealed when fixation nystagmus is abolished by the cessation of fixation.

Principles and technique of electronystagmography are briefly outlined by Dieterle and Monnier. Recording of horizontal movements of the eyeballs is best obtained by placing electrodes at the inferior margin of the orbit, symmetrically in reference to the orbital axis, which is somewhat lateral to the visual axis. Nystagmography allows an exact analysis of the characters of optokinetic and vestibular nystagmus, either normal or pathological. By contemporary recording of optokinetic nystagmus from both eyeballs (the one being the fixing eye, the other eye being closed) it was possible to state that the nystagmic beat has a greater amplitude in the fixing eye and, furthermore, that it reaches its maximum amplitude when the eye is fixing a central point. In congenital nystagmus, it has been observed that frequency and amplitude of nystagmic beats change in connection with the position of the fixing eye, which would point to the fact that the activity of the automatic center, such as that of nystagmus, is influenced by afferent proprioceptive excitations coming from the eye muscles. Nystagmography permits one also to give an explanation of compensatory torticollis; there is a position of the eye where nystagmus has the minimum of amplitude and the gaze is relatively steady; nystagmography enables us to predict the position of the head which the patient will take in order to obtain the best stability of gaze. Analogously, when congenital nystagmus is present, squint is frequently observed on account of a loss of the binocular vision. In these cases, nystagmography enables us to state that the position of the eyeball for which the amplitude of nystagmus reaches its minimum is different in the two eyes. Electro-oculography has enabled the authors to determine the optimal conditions for the electrophysiological analysis of the nystagmus of each eye, the fixing as well as the closed eye (consensual nystagmus). It is possible, for instance, to analyze the variations of the optokinetic nystagmus with various positions of gaze and to record the vestibular nystagmus. This is of practical interest for otocompensatory torticollis and squint.

A description is given by Henriksson of a method for electrically analyzing the movements of the eyes in nystagmus. According to this method, the speed of the slow phase is given directly. According to the author the nystagmographic method presented in this paper has several advantages: 1. The nystagmus curve immediately gives an amplitude corresponding to the speed of the slow phase of nystagmus. 2. The tracing can be compressed and thereby provides a good survey even of the curve of nystagmus of long duration. 3. The derived curve readily reveals nystagmus strokes even when very weak nystagmus is recorded.

Serial examinations were carried out by Kirstein in test persons with normal vision and normal hearing, both with the eyes open and with the eyes closed. It was proved that during electronystagmography after closure of the eyes disturbances occur, which may be so marked that the recording and assessment of vestibular nystagmus becomes difficult or even impossible.

The theoretical basis of nystagmography is discussed by Pivotti and Lucarelli; the corneoretinal potential is used. An accurate review is presented of all the factors on which nystagmographic study depends—the intensity of the currents which follow ocular movements, the characteristics of the electrodes and of the elective points of derivation, the apparatus in which the electrodes are contained, which the authors have personally devised and which is easy to use and very accurate. Data concerning the preamplificator are extensively discussed, with a description of its scheme; it has been included in order to make possible a more exact registration of a phenomenon so variable in frequency and intensity as ocular nystagmus can be. The amplificator is connected with an ECG apparatus. Reports are

presented on a number of nystagmographic curves registered under various conditions (vestibular and optokinetic stimulation, etc.).

The value of electronystagmography as a way to examine the vestibular organ is reported by Reich. Rotatory nystagmus, however, cannot be registered, and all registrations show a certain distortion. Electronystagmography gives us information about vestibular function through the medium of ocular movements. It cannot possibly indicate whether what is registered is of peripheral, central, or ocular origin. To solve this problem, the author suggests the use of electroencephalography, the measurements of electric effects in the neighborhood of the labyrinth capsule, and electrocardiography.

Recording of the skin resistance in thermal and rotatory stimulation of the labyrinth are reported by Behr, Preber, and Silfverskiöld. The present investigations were designed, by graphic recording of the variations in skin resistance, to throw further light on the nausea associated with sweating, etc., that may attend rotatory and thermal vestibular stimulation. A specially designed apparatus was employed for recording. About 50 patients were investigated. Vertigo induced by slight rotatory stimulation (cupulometry) was sometimes accompanied by a fall in skin resistance that was difficult to distinguish from nonspecific falls due to emotional factors. Thermal stimulation often, after a latent period of two or three minutes, produced a pronounced fall in skin resistance coincidentally with nausea. The fall sometimes appeared without demonstrable sweating or other objective signs of nausea. The causes of this evidently characteristic fall in skin resistance are briefly discussed. The method employed here seems to be serviceable as a simple means of recording vegetative vestibular reactions.

On the basis of the fact that definite relationships have been demonstrated between labyrinthine and cochlear function on the one hand and variations of the intracranial pressure on the other, a study was made by Maffei of 20 subjects who had undergone unilateral or bilateral resection of the lymph nodes of the neck with ligation of the jugular vein. In the first months after the operation there was often an altered reactivity to vestibular stimulation tests, with variable intensity and type. Young people showed the most marked variations, which were, however, also more transient, and old people, longer-lasting changes. There was a certain parallelism between the increase of the C. S. F. pressure and the irregular vestibular reactions to the labyrinthine stimulation tests. The late result was very often hypoexcitability, never, however, accompanied by marked subjective vestibular disturbances. As regards the anterior labyrinth, there was never any evidence of significant deviations from the normal. In conclusion, it is stated that after the operations mentioned the variations of C. S. F. pressure are transmitted also to the labyrinthine fluids; this pressure disturbance manifests itself in a change of the response to vestibular stimulation tests.

In some cases of chronic otitis, the changes of the inner ear are so minute that they cannot be detected by usual methods of examination. They can be discovered, however, after the exclusion of the depressive influence of the C. N. S. Markova eliminates the influence of the C. N. S. by using chloral hydrate in 20 healthy people, in 60 cases of chronic otitis without affection of the vestibular apparatus, and in 70 cases of chronic otitis, complaining of vertigo. In this last group he found that, after elimination of the function of the C. N. S., it is possible to register changes in the reaction of the vestibular apparatus to irritation, which could not be detected before. In six cases with abolition of labyrinth function and in five cases after radical operation, caloric nystagmus appeared after the application of chloral hydrate. In this group, nystagmus after rotation was also prolonged after the administration of chloral hydrate.

Anatomy of the Ear

Beginning with invertebrates (coelenterates, arthropods, insects) and continuing with the classes of vertebrates, it is shown, by Gray, how the organs for the sense of position and movements in space and of sound have gradually developed. Good illustrations and diagrams accompany the text, which gives a useful survey written especially for otologists.

Pregnant albino rats were exposed to x-rays at midterm, and their litter were examined histologically close to term. The aim of Kelemen was to compare the radiation effect, as shown by malformation and hemorrhage, with similar, previously described human conditions. The auditory, vestibular, and olfactory end-organs were found to be structurally intact, in appropriate developmental stages, amid extensive hemorrhages in their vicinity and against a background of severe, radiation-induced endocranial deformities. The teratological damage as encountered here can be typed as environmental, not caused by an abnormal condition of the effective end-organ itself. The functional effect would be, probably, a total loss after destruction in the central pathways. Moreover, it is known that organs appearing intact immediately after an insult may still degenerate, and vascular radiation defect may become manifest later in the form of a birth injury or during any phase of postnatal life.

Marullo injected pyruvic acid in a dilution of 1:6 and in doses of 0.25, 0.05, and 0.1 ml. into the yolk sac of eggs of Leghorn chicks. The embryos of 11, 13, and 16 days showed histological lesions of the epithelium, similar to those of genuine neuroepithelial degeneration, and degenerative lesions of the eighth nerve with interstitial edema dissociating the neurons of the acoustic and vestibular ganglions, chromatolysis, etc.

On the basis of the fact that embryonal tissues normally respire and of a great many data derived from teratological experiments with low-oxygen atmospheres, it is concluded by Marullo that hypoxia should be regarded as one of the possible causes of blastophoric malformations. Several investigators have been able to demonstrate that respiration is intensified during preceding embryonal division, from which it can be concluded that oxygen is necessary for cellular multiplication and for embryonal differentiation. It is precisely the most highly differentiated structures which require the highest amounts of oxygen; the nerve structures suffer most from the relative oxygen deficiency during development. The purpose of the present study was to determine the consequences of atmospheric hypotension on auricular organogenesis. Exposure of chick eggs during the whole period of development to atmospheric hypotension (values between 20 and 50 cm. Hg) proved to have no malformative effects on embryonal auricular organogenesis. There were only slight developmental retardations, which, in any case, were in accordance with a general hypoplasia shown by the embryos. It can, therefore, be concluded that the embryo is reasonably resistant to a condition of chronic hypoxia to which the egg is exposed from the beginning of the development. Although the reduction of the oxidation processes leads to hypoplasia of the embryo, the differentiation of the structures and organs takes place as usual. There is a much greater danger of blastophoric malformations in case of sudden oxygen deficiency, even when it is of transient nature, because the embryo has not then the chance of adapting itself as in case of chronic hypoxia.

A study with the phase-contrast microscope was undertaken of the spiral organ of the guinea pig, by Vago. The first important finding was the high degree of cytoplasm-absorbing capacity which characterizes the supporting cells and, somewhat less, the acoustic cells; the cytoplasm of an isolated supportive element tends to accumulate in the form of drops, made possible by the resistant fibrillar formations it contains. Another striking finding was the ease with which the elements can be separated from one another, except in the apical part, where

the cuticular formations of the acoustic cells, of the pillars, and of the Deiters cells are closely connected, presumably by a cementing substance. From these findings it may be concluded that the mechanical importance, for the maintenance of the shape of the spiral organ as a whole and of its separate elements, lies partly in the pressure exerted by the fluid which fills the membranous cochlear duct and the spaces in Corti's organ (Corti's duct, Nuel's space). Examination in fresh preparations of the acoustic cells revealed that these cells adhere firmly to each other with their heads, where the cuticular membrane is situated, whereas they may easily be detached from each other along the body and at their ends from the corresponding notch of the Deiters cells. The cuticular membrane is not of uniform thickness; its lower surface is irregular; its upper surface, smooth; on the latter, the hairs are implanted. The acoustic hairs appear to be implanted in the central zone of the cuticular membrane, following a U-shaped line in the outer cells and a straight line in the inner cells. The cytoplasm looks homogeneous and grayish yellow, with a large number of small granules, which are particularly numerous in the subcuticular region, which probably constitute Hensen's body, and in the fundus of the cell, which probably constitute the organ of Retzius. No vacuoles are present. The nucleus, which is perfectly round, lies in the fundus of the cell, directly over the Retzius body. The chromatin is clotted into lumps of various sizes. When the size of the cells was determined, the values in fresh preparations were found to be slightly higher than those in fixed preparations. Half an hour after the initial observation of the fresh preparation, a number of cellular alterations due to postmortem phenomena become visible in the acoustic cells, the most striking of which is the gradual formation of vacuoles in the cytoplasm; the vacuoles become progressively larger and finally occupy the whole cell. The isolated Deiters cells observed in fresh preparation are seen to hang roughly together; the notch which connects them with the acoustic cell is not very much bundled together, following one single side of the cellular body, and in axial position at the neck. The nucleus is identical with that of the acoustic cell. The neck of the Deiters cell runs alongside the body of the acoustic cell but does not adhere to it. The phalanges are composed of homogeneous material. The postmortem changes consist here also mainly in vacuolation and imbibition of cytoplasm. The pillar cells, studied in fresh preparations, lose their normal coherence and appear bulb-shaped; the postmortem changes are the same as mentioned above. Study of fresh preparations did not allow of differentiation between Hensen's cells and Claudius cells, owing to the tendency, already referred to, of all these cells to assume a globar shape. In fresh preparations the various elements which compose the reticular membrane presented no filamentous or granular structure and should therefore be regarded as formations of homogeneous, structureless substance.

Engström has carried out a light and electron-microscopic analysis of the basilar membrane. There is a discussion of the different layers within the membrane, and the question is raised whether the pronounced difference in these layers corresponds to important functional differences and different mechanical properties.

The stria vascularis of the guinea pig was studied by light and electronic microscopy by Engström, Sjöstrand, and Spoendlin. The so-called stria epithelium consists of superficial chromophil and deeper chromophobic cells. The first type of cell has an electron-opaque cytoplasm, with numerous relatively large mitochondria. The second type contains a much brighter cytoplasm, with numerous pigment particles. Tendrils of both cell types make contact with the blood capillaries of the stria. The authors raise the question whether the dark cells only are of epithelial origin whereas the bright cells are derived from the mesenchyma.

Tsuiki, Watanabe, and Kamioka have fairly succeeded in grasping the living state of the various parts of the membranous labyrinth by use of a phase-contrast microscope for their examination. These experiments have the advantage that they can rid themselves of the fixation method and take living cells as their object. Also, the time required for the preparation of microscopic slides is cut down to almost nothing. They could in this way follow up the so-called postmortem degenerations of Corti's organ and arrive at many valuable observations connected with the disintegration, swelling, and bubble formation occurring therein. The various cells in the ear labyrinth showed diverse reactions on application of several chemicals and medicaments, such as swelling, contraction, and stoppage of granular movement.

X-ray diffraction technique was used to analyze the composition of statoconia and statoliths from 14 different vertebrate species. The crystallographic structure varied from species to species. Studies by Carlström and Engström, with the electron microscope, showed that the human statoconia might be composed of small hexagonal units of calcite arranged in a well-ordered pattern; similar pictures can be obtained from crushed crystalline calcite. The regular form and the x-ray crystallographic analysis of single statoconia show the latter as single crystals. The hexagonal platelets have to be so well aligned that the whole statoconium behaves like a single crystal.

The cochlea of bats caught during hibernation was found by Plotz and Perlman to be suitable for detailed morphological and histochemical study after fixation by freezing and drying. The histochemical methods included the Hotchkiss procedure (with solubility tests), metachromasia, and staining procedures for alkaline phosphatase, neutral fat, and plasmalogen. The fibers of the tectorial membrane stained deeply with the Hotchkiss method, but no plasmalogen was found there. A marginal network, reacting histochemically like the fibers of the membrane, was seen in nearly all sections. Hotchkiss-positive granules exhibiting metachromasia were demonstrated in the pink-stained cytoplasm of the stria epithelium. The Hotchkiss-positive material in the stria cells was not extractable with 0.1 N of hydrochloric acid, buffers of various pH, and saliva. The cytoplasm of the cells revealed a positive reaction for plasmalogen. Alkaline-phosphatase activity was found in the basal part of the stria vascularis. Hotchkiss-positive granules exhibiting metachromasia were found in the cytoplasm of the epithelial cells of Reissner's membrane. The membrana propria stained intensely by the periodic acid-leukofuchsin reaction and was not extractable by the reagents employed. The article is illustrated with some very fine photomicrographs.

Vilstrup, Jensen, and Koefoed found that the labyrinthine fluids have the rheological property of flow elasticity accompanied by electrical effects, flow potentials. Differences between perilymph and endolymph were measured as to the numerical values of the potentials, to elastic recoil, and to conductivity. It is possible that the hydrops-like dilatation of the endolymphatic sac in Ménière's disease is an osmotic consequence of a depolymerization of the hyaluronic acid. Flow potentials are a new departure, and their discovery opens strategic questions concerning the mechanism of the inner ear and will intensify the interest in the electrolytic composition of the labyrinthine fluids.

The labyrinthine fluids of various animals, cat, hen, and rabbit, were studied by Casorati and Posarelli under the electron microscope. The presence of typical tree-shaped formations, attributable to elongated macromolecules, which very often support the crystals, was detected. This aspect is reminiscent of that formed by the elongated molecules of synthetic polyanions with colloidal particles of caolin. No substantial differences were found between the labyrinth fluids of the various animals.

Morphological characteristics of human labyrinthine fluids as seen by the electronic microscope are reported by Casorati. En-

dolymph is formed with elongated proteinic macromolecules, crystals, and roundish formations of uncertain interpretation. The perilymph shows only protein chains and cubic and prismatic crystals.

The proteins of the perilymph of the cat from the quantitative and qualitative point of view are analyzed by Casorati and Crifo, The refractometric quantitative analyses appear to show the presence of a considerably greater quantity of proteins in the perilymph than had been believed until the present: total proteins, 1.65μg. per 100 ml., i. e., 1.37μg. globulin and 0.28μg. albumin. Research with electrophoresis has enabled the perilymphatic proteins to be fractioned; these appear to consist mostly of globulins, particularly those of the γ-type, and the proportion of albumin is relatively smaller.

The following amino acids have been detected, by Antonini, Casorati, and Crifo, in the perilymph from horses, by filter paper chromatography: aspartic acid, phosphocolamine, glycine, glutamic acid, serine, taurine, threonine, alanine, glutamine, lysine, arginine, α-aminobutyric acid, proline, valine, leucine, tyrosine, phenylalanine, and cystine. A comparative study of chromatograms obtained from blood, perilymph, and liquor showed that the perilymph is much more similar to the blood than to the cerebrospinal fluid either for qualitative or for quantitative content of amino acids.

A report is presented by Coassolo of studies of the ultrastructural morphological characteristics and the physical properties of otoliths. These appear as crystalloid formations with a low atomic weight, sometimes hexagonal, sometimes multiple, and composed of countless microcrystals. The inorganic nature of these crystals could be proved by refraction tests. Other crystalloid formations with high atomic weights, hexagonal or rhombohedric, which the author calls paracrystals, are found not only in the utricular and saccular macula but also in the cupula of the semicircular canals. These paracrystals are said to be of organic nature.

The development of the otoliths is described by Lyon. In alcohol-fixed material the first appearance of the otoliths (O) was found in the 14½-day embryo as a protein precipitate. After formalin and Bouin fixation the first sign of O could be found only at 16½ days and 2 days after birth, respectively, but after alcohol the localization of the O material was unreliable. At 16½ days, the O consisted of a gelatinous layer, surmounted by a layer of birefringent calcareous granules with an organic matrix, containing PAS-positive, amylase-resistant polysaccharide, glycogen, and alkaline phosphatase. The macular cells showed also phosphatase activity.

Otoliths of men, 24 rabbits, 32 guinea pigs, 3 rats, frogs, and mackerels were examined by Sasaki and Miyata by means of the Meigen reaction, and the electronic microscope, and the x-ray spectroscope. Solutions of sodium bicarbonate and sodium citrate are not able to dissolve otoliths. The theory that solutions of sodium bicarbonate can dissolve them is proved erroneous by histological findings. Otoliths of men, guinea pigs, and rats consist of Calcit ($CaCO_3$, calcium carbonate). Otoliths of frogs and mackerels consist of Aragonit ($CaCO_3+H_2O$).

Seventy unfixed chicken embryos were used by Savin. In the endolymphatic sac of a 5-day-old embryo, crystals of calcium carbonate were found. The amount of crystals decreased during development; in the newly born chick only an extremely small white-colored mass is still present. A crystallographic study of the crystals showed that they are of the same nature as the otoliths of the utriculus, sacculus, and lagena; the only difference is their much smaller size.

Cochlear Physiology

Pathological physiology of hearing disorders is discussed by Bornschein. The magnitude of the cochlear (hair cells) microphonic potential depends on the physical intensity and the physiological efficiency of the hair-cell stimulation. In

conduction deafness the stimulation energy working on the hair cells is reduced. In perception deafness the transformation of the physical stimulation into the physiological response is disturbed. The transmission of the mechanic energy of the sound is dependent on the mechanic properties of the middle and internal ear. Specific graphs are found by means of charting the amplitude of the potential against the intensity of stimulation. A double logarithmic system of coordinates is used. When a perforation in the drum is performed, the graph of the intensity is altered horizontally in the direction of the higher tone intensities. The loss can be compensated by increase of the stimulation intensity. When the transformation process is disturbed by anoxia, the graph of the intensity is altered vertically in the direction of the lower potential amplitudes. The loss cannot be compensated completely. The alteration of the graphs, i. e., the loss of hearing, can be read in decibels. Analysis of mixed deafness is more accurately done by this method. The transmission of the high frequencies takes place directly through bone conduction, with increasing loss of energy. Middle ear disorders, therefore, lead to a hearing for the lower frequencies. After a radical mastoidectomy the hearing loss is equal for all frequencies. There are two components: (1) deafness for the low tones, since the mechanical transformation does not occur, and (2) deafness for the high tones owing to a reduction of masses—ossicles—normally coupled with the perilymph of the scala vestibuli. In perception deafness, the process which transforms the physical stimulation into physiological response is disturbed. Since the electric energy produced by the hair cells is larger than the mechanical energy of the sound, the transformation process includes a mechanism of amplification, where chemically bound energy is freed. This metabolism needs oxygen. Anoxia of more than five minutes does not allow complete recovery. The primary neuron of the hearing pathway is more easily damaged in anoxia than the hair cells. The definite hearing loss is therefore more extensive than the microphonic potential shows. A functional and histologically proved damage of the hair cells is found with quinine, streptomycin, neomycin, and chenopodium oil. The analysis of the sound within the inner ear is based upon the fact that the curvature stiffness of the basal membrane is changing continuously from the base to the apex. The lowest frequencies advance most into the apex. Disharmony of sounds occurs when a very loud sound is given to one ear, followed by binaural sound comparison. In the pretreated ear the sound will be lower or higher than in the other one. It is explained by fatigue and the known swinging form of the basal membrane. This phenomenon is present in perception deafness at the beginning of the damaged frequencies. However, the diplacusis binauralis after fenestration (all frequencies sound higher) is due to a change of mechanical factors. Specific for perception deafness is the recruitment phenomenon. Loudness difference between the good and the diseased ear is lessening with increase of intensity until the loudness is the same bilaterally with very high intensities.

The physical properties of the organ of Corti are discussed by Bekesy with reference to model experiments. In the tongue frequency determinator, whose elements are coupled by means of a rubber band, vibrations produce migrating waves which invariably move from the more inflexible to the softer part of the original spot of the driving power. The basilar membrane of the organ of Corti behaves in the same manner; the waves also invariably move toward the helicotrema even if the driving power is experimentally placed elsewhere than at the round window. All these wandering waves have an amplitude showing a smooth rise to a maximum from the stapes on and then showing a fairly steep fall. The maximum shifts with increasing

frequency to the stapes and with decreasing frequency to the helicotrema. The organ of Corti on the basilar membrane follows these wave movements but shows certain characteristics of its own. The Hensen cells, for instance, vibrate from the stapes to the level of maximal amplitude; in a radial direction at the maximum an up-and-down movement occurs and the longitudinal axis changes toward the helicotrema. Further experiments were made regarding the problem of frequency analysis, which is still unsolved. Since inhibitory phenomena are of great importance in this respect, the tactile sense of the skin was used, so to speak, as a biological model of the organ of Corti. An enlarged cochlear model was designed in such a manner as to make it possible to place the anterior arm on the vibrating membrane, thus determining vibrations by means of the tactile sense. The vibratory maximum was always experienced as sharply circumscribed, although the entire membrane was in motion. With increasing frequency it shifted to the foramen ovale, and with decreasing frequency, to the helicotrema.

Continuing with his extraordinary experiments on the physiology of the cochlea, Bekesy investigates further the formation of traveling waves in a model having properties mechanically analogous to the cochlear partition. The paradoxical movements of a wave traveling toward the source is the phenomenon which the author has observed only in the cochlear mechanism. By use of the mechanical model, an explanation is given for the observation that waves may travel toward the point of maximum driving pressure as well as away from it. By changing the position of the various mechanical elements on the model, attempts were made to explain the various types of waves observed.

Engström discovered that at least the majority of the outer hair cells in the organ of Corti are provided with intracellular structures that would be well suited to function as an electromechanical transducer. It is now known that the mechanical distortion of polarized molecular layers may transform mechanical energy into electrical energy. It is observed that the supranuclear portion of many outer hair cells is provided with a large structure of a membranous nature with well-defined layers, which forms a suitable basis for further discussion on the anatomical substrate for the release of cochlear potentials.

Measurements in histological specimens, according to Hilding, have shown that the basilar membrane narrows in its apical portion, whereas throughout the rest of its length it widens progressively from the base toward the apex. Since place theories of hearing are based partly on the graduated size of the parts, the narrowing of the basilar membrane in the apical turn appears to be inconsistent with any place theory. Observation of the motion of the cochlear partition in the final half turn through a cochlear window showed that the width of the moving portion of this partition increases progressively almost all the way to the tip of the organ of Corti. The limbus and part of the lamina spiralis could be seen to swing with the basilar membrane, although to less extent. The amplitude of movement increased along the basilar membrane by virtue of the membranous portion of the helicotrema until the tip was reached. These experiments show that the width of the basilar membrane in the apical half turn does not delimit the dimensions of the portion of the cochlear partition which take part in motion.

When the anatomical structure of a sensory organ of the auditory apparatus becomes changed as the consequence of some disease, its functional condition will remain deteriorated to a degree proportional to the extent of the alteration itself. When, however, the alterations pass a certain point, the functional deficiency is no longer susceptible to deterioration, even when the lesion is aggravated further. Fiori Ratti and Marullo name this situation the critical level of functional invalidation. The functional deficiency corresponds to that which

would be caused by the absence of the structure in question plus a certain parasite effect due to the fact that the functionally inefficient structure still remains in situ. The clinical application of this concept is of considerable interest, especially with regard to functional surgical treatment of ear affections.

Llaneza Rodriguez discusses the concept of Tasaki, Davis, and Legouix, according to which two simultaneous sounds are propagated along the cochlear turns with different velocities, with one of them suffering retardation in its course. He does not accept the possibility of separation of the two frequencies, which, on the contrary, blend. The two components fuse into a single sound, the latter containing some characteristics of both but being different from each of the original two. The basis of his concept is the law according to which the velocity of the propagation of the vibrations is independent of the source of the sound and independent of the vibrations; the propagation depends exclusively on the medium to be penetrated.

Any vibratory device emitting fast waves above a specific range may produce functional and structural changes in the hearing organ, reports Angeluscheff. Exposing the ear of animals to ultrasonics of 38,000 cps. for 30 minutes daily for seven days has shown disruption of Corti's organ, broken pillar cells, hemorrhages in the scala tympani, shrunken nerve and ganglionic cells, and new bony tissue of bluish discoloration formed from the periosteum and endosteum. This bony tissue may penetrate the structure and cause fixation of the stapes. In everyday life hearing is exposed to ultrasonic impacts. One or two microwatts of energy is to be regarded as sufficient to initiate migration of ions, which is the chief process in this phenomenon. Modern statistics show the great number of people suffering from deafness, while sound amplifiers are also increasing in number.

Submicroscopic micropipette electrodes were introduced through the opened bulla and a burr hole between the posterior semicircular canal and the scala tympani of the cochlea and into the cochlear nucleus in the medulla of guinea pigs, by Tasaki and Davis. From about 80 elements, unitary electrical discharges were recorded; their response to sound stimuli (tone pips and pure tones), applied through the opening in the bulla, was investigated. Microphonic and whole-nerve responses were recorded simultaneously by a nichrome-steel wire inserted into the scala vestibuli of the basal turn of the cochlea. Single-unit responses of the cochlear nucleus were exclusively positive spikes less than 1 msec. in duration. Spontaneously discharging units were often encountered. The rate of spontaneous discharge could be increased by appropriate acoustic stimulation, but the discharge could never be inhibited. Many units responded synchronously with the peak of the whole-nerve response, indicating activity in primary neurons. Discharges in secondary and higher-order neurons having a longer latency were also met. Response areas for acoustic stimuli of varying frequency could be mapped in favorable cases. The responding elements showed sharp frequency cut-offs at the upper boundary of their response areas, whereas on the low-frequency side the sensitivity decreased only gradually.

In guinea pigs, Gisselsson measured the electric potentials of the endolymph and perilymph fluid. As soon as the microelectrode perforates Reissner's membrane a positive potential of 40-75 mv. is found. In the immediate neighborhood of Reissner's membrane (50μ to 75μ) it becomes negative (-75 to -100 mv.). In Corti's organ the potentials fall to 0. Oxygen absence has a great influence on these potentials. Cochlear potentials are much greater in the endolymph than in the perilymph.

Tones of 500, 1,000, and 2,000 cps were conducted into the external auditory meatus of guinea pigs and cats. The cochlear microphonics were picked up from the labyrinth capsule and photographed on an

oscilloscope. When the round window was firmly closed by dental cement, the cochlear microphonic potentials appeared markedly reduced. After removal of the cement the potentials approximated the original magnitudes. These experiments show that the closure of the round window in the case of a normal drum and ossicular chain produces a marked hearing loss. The phenomenon according to Gisselsson and Richter has no connection with the improvement of hearing by screening off the round window in a radical cavity.

Livan, criticizing the usefulness of tests proposed up to now for the clinical evaluation of the function of the round window, concludes that there does not yet exist any instrumental means which may achieve such a purpose.

In a recording from intact human skull through epicranial electrodes placed on the acoustic region, smooth currents were registered, by Kohler and Wegener, which greatly resembled those obtained from the auditory cortex of the cat under sound stimulation. As in the cat, this type of stimulation failed to block the normal electrical rhythm of the auditory cortex. After repetition of stimuli the smooth current was reduced in size, and after prolonged experimentation very slow waves sometimes appeared in the absence of stimuli.

Dogs were subjected, by Faure and Portmann, to auditory stimulations, varied elementary (click, tap, tone pipe) complex, auditory-visual association, and sound film projections. Simultaneously an electrode driven into the middle part of the hippocampus and a surface electrode made it possible to analyze the responses obtained. Responses to simple stimulations are detained in the hippocampus by comparison with the cortical surface. Complex noises bring about accidents of a longer latency, an effect "on," and effect "off," and modifications of the morphology of the responses. The complex situations show that the auditory complex situations force themselves on and inhibit the phenomena obtained by visual afferences in most cases. On the other hand, the emotional and postural state objectivated by electromyogram is strongly influenced by the sensorial afferences.

In 64 guinea pigs under urethan anesthesia, the effect of the temperature on the cochlear microphone potential (MP) derived from the dorsolateral segment of the basal convolution was investigated by Bornschein and Krejci. The decrease in potential caused by ischemic anoxia (aortic compression) shows a different course in hypothermic animals from that in animals at normal body temperature. Orienting experiments suggest an analogous prolongation of the resuscitation time for this potential in the case of hypothermia. Brief local cooling of the cochlea in the ventral region of the second to fourth convolutions caused complete reversible disappearance of the MP, including the anoxia-resistant component (MP of the second order). During cochlear rewarming the MP shows transient pulsatory modulation of amplitudes, asymmetrical nonlinear serrations, and phase shifts exceeding 90 degrees. By thermoelectrical control determinations it was demonstrated that disappearance of the MP is not caused by extension of the hypothermia to the basal cochlear convolution. The question arises as to the extent to which the effects observed can be attributed to a change in the temperature-dependent mechanical constant.

Furstenberg reviews the nerve supply of the blood vessels of the head. Basic physiologic principles of vascular behavior are portrayed, and their clinical application to symptoms and diseases of the inner ear is discussed. An interesting aspect presented is the role of hypertension in the etiology of nerve deafness, with improvement in the hearing on reduction of the blood pressure. The evidence is given for assuming that Ménière's disease is due to vascular alterations. The physiologic basis for the various types of nystagmus is presented. The action of vasodilators, especially atropine, in Ménière's disease is described. Also dis-

cussed is the syndrome of sudden deafness, severe tinnitus, and vertigo and nausea; this entity is best explained on a vascular basis.

The behavior of the small vessels during allergic reactions was studied by Irwin, Weille, and Burrage on an undetermined number of guinea pigs and rats. The observations were carried out on the small vessels of the spiral ligament of the cochlea, of the lungs, and of the liver. Some interesting data on the small vessels of the cochlea were described. When the animals died two minutes after the beginning of the anaphylactic shock, the arterioles and the arteriovenous anastomoses of the spiral ligament of the cochlea contracted and both networks of capillaries became less evident. When the animals died after 5-10 minutes, beside the above alterations, the formation of small thrombi was observed. When the animals survived, there was an almost complete "restitutio ad integrum" within 15 minutes. Similar alterations were observed in the vessels of the lungs and of the liver. Also during the histamine shock the small vessels underwent similar changes.

Perlman and Kimura have amplified the now familiar knowledge of the anatomy and hemodynamics of the circulation in the cochlea by the description of further detail. The vasomotor control of these vessels was investigated, and the stimulation of the sympathetic trunk and superior cervical ganglion produced no visible changes in the vessels or the blood flow. A number of experimental and pathological states, including anaphylaxis, were reexamined. An important conclusion is the possibility of localized pathology with vascular lesions in the stria vascularis, although under physiological conditions the circulation is remarkably stable.

Perlman and Kimura describe the blood flow in the spiral ligament of a guinea pig exposed by fenestration, as seen under the microscope. They describe the radiating arterioles of the spiral ligament, the capillary bed, and the collecting venules. In addition, the now familiar arteriovenous arcade is demonstrated. They believe the vascular bed is remarkably stable, there being little evidence of spontaneous vasomotion.

The blood circulation of the spiral ligament was studied by fenestration of the cochlea over the spiral ligament in living guinea pigs, reports Weille. The trauma was so little that an approximation of normal blood flow was retained. Arteriovenous arcades were found in the spiral ligament carrying arterial blood directly to a collecting venule, without intermediation of the capillary bed. The arcades are wider than capillaries and thicker walled. It is likely that a major function of these arteriovenous shunts in warm-blooded animals is heat transfer to the tissue, especially a peripheral one. They are not adequate, as capillaries are, for the diffusion, filtration, and reabsorption required in tissue nutrition. Since heat or cold are easily transmitted from the external environment to the inner ear, these arteriovenous arcades appear to provide a superb physiological heat-regulating system for the cochlea. It is, furthermore, suggested that these arcades function in part to mobilize blood rapidly to an area of greater usefulness, e. g., the capillaries of the stria vascularis, in accord with the need for the regulation of the secretion of endolymph.

Permeability of living membranes plays a very important role, mainly protective and nutritional, in biology. Zaiko refers to his earlier experiments on vascular permeability of the eye, in which he examined the differences of chemical composition between the aqueous from the anterior chamber of the eye and the blood. He concluded, "Haemato-ophthalmic barrier depends almost wholly on the permeability of the walls of capillary blood vessels." Now, he set out to compare the vascular permeability of the inner ear with the eye. The greatest difficulty in determining the chemical composition of the fluids of the inner ear lies in the small quantity obtainable in experimental conditions. This difficulty can be

largely overcome by use of the method of "tagged atoms." The author carried out experiments on cats and dogs, giving them intraperitoneal injections of a certain amount of a solution of natrium phosphate ($Na_2HP^{32}O_4$) containing radioactive phosphorus (P^{32}). After an hour, each animal was killed, the bulla was opened, and the labyrinthine fluid was aspirated through the round-window membrane. The fluid thus obtained was deposited on standard filter paper, weighed, and the activity of such a preparation measured. At the same time, concentration of radioactive phosphorus in the aqueous content of the anterior chamber of the eye, cerebrospinal fluid, and blood was determined in the same way. In these experimental conditions, the level of P^{32} in the labyrinthine fluids, C. S. F., and the aqueous was found to be, on the average, 5-8 and, sometimes, even 25 times lower than in the blood. In the author's opinion, the existence of this "haemato-labyrinthine barrier" plays a very important role in keeping up the steady chemical composition of the labyrinthine fluids, necessary for the exact functioning of the inner ear. The animals in the next series of experiments (cats) were submitted to a strong acoustic stimulus (electric bell) for 15 minutes after injection of the radioactive phosphate. The level of P^{32} in the labyrinthine fluids and also in the aqueous was then found to be markedly higher than in the first series of experiments (no figures are given). In one experiment, the level of P^{32} in the labyrinthine fluid was still 1.8 times higher than in the aqueous. The author believes to have proved by this experiment that the functional state of the organ influences its vascular permeability. In the last group of experiments, intracranial section of one trigeminal nerve was performed. In these animals (cats) the levels of P^{32} in the labyrinthine fluid and in the aqueous rose considerably, in comparison with the control animals not operated on. But, on the side operated on, the levels of P^{32} were much higher than on the opposite side in almost all animals. These results seem to indicate a reflex influence of the nervous system on the vascular permeability of the inner ear and the eye.

Asphyxia was induced in guinea pigs and cats by Fernandez in order to determine the survival time, recovery time, and revival time for cochlear microphonics (CM) and action potentials (AP). Two phases of CM are identified. On the basis of the rapid decline of the fast phase a high metabolic rate of the hair cells is inferred. Lack of dependence of this decline upon stimulus intensity suggests equal oxygen requirements among the hair-cell population. The short survival time of AP supports the view that the cochlear nerve has a high metabolic rate, close to that of the C. N. S.; definite dependence of the survival time of AP upon stimulus intensity indicates that there are groups of fibers with different oxygen requirements. Short recovery time of CM reflects the rapid rate of physicochemical reconstruction of the hair. Occasionally there is overshooting. Repetitive asphyxia usually produces cumulative depression, probably due to irreversible changes. Recovery time of AP is of the same order of magnitude as that of the C. N. S. Recovery time of AP varies, as does survival time, with stimulus intensity. This represents different rates of physicochemical reconstruction among the primary cochlear neuron population. AP recovers fully, and as a rule overshooting, at times as high as 75% over normal, occurs. Lower threshold fibers are characteristically the most sensitive to repeated asphyxia. Revival time for asphyxia cannot be determined because the animals die in seven minutes.

A histological investigation was carried out, by Maffei and Marcucci, of the changes of the inner ear in guinea pigs in conditions of anoxic anoxia. A description is given of the apparatus used for these experiments, and a report is given of the results in 12 guinea pigs, subdivided into groups and exposed to simulated altitudes of 5,000, 7,000, and of 9,000 meters, re-

spectively, for periods of two hours, and this 15 times. On the whole it may be said that the auricular structure was affected to a considerable degree, with the appearance of lesions of degenerative type, varying with the simulated altitude. The vascular and connective elements were earlier and to a severer degree affected than the ciliated cells, which exhibited on the whole a greater resistance against the noxa of anoxia. The earliest and worst lesions were found in the basal gyri of the cochlea, where the highest tones find their acoustic projection.

Cochlear microphonics were found by Tonndorf, Hyde, and Brogan to be affected by oxygen deprivation of moderate degrees; this effect depends upon the degree of oxygen deprivation and on the magnitude of simultaneous exposure to sound. A normal oxygen supply was reintroduced at the termination of sound exposure; the rate of recovery of the cochlear microphonics was faster than after sound exposure without return to normal oxygen supply. This can be taken as evidence that during production of cochlear microphonics and in gross proportion to the magnitude of sound exposure electrical energy is drained from the cochlea. Cochlear microphonics, as produced in the organ of Corti, are not mere by-products but represent the essential link between mechanical stimulation of the cochlea and nervous excitation.

The maximum strength of the tympanic muscles was studied by Wever, Vernon, and Lawrence. On anesthetized and decerebrated cats the middle ear structures were exposed and a fine silk thread was tied to the tendon of the muscles and connected with a strain gauge. The muscles were stimulated to maximum contraction by electric currents. The strain gauge consisted of four resistance wires in a Wheatstone-bridge arrangement. Large individual differences were found, with 3.5 g for the tensor tympani when connected to the ossicular chain and with 4.4 g when freed of the chain. For the stapedius, the means were 1.6 g for both conditions. Damping of vibratory motions of the chain was found to be the primary action of the muscles, and their imposing tension is only a secondary effect. Tensions imparted by the two muscles are partly in opposition, with a resulting protection of the inner ear against overstimulation by sounds with a minimum of displacement of the ossicular mechanism from its normal operating position.

Experiments were carried out by Wever and Vernon on the cat to show the changes in sound transmission by the ear caused by a reflex contraction of the tensor tympani and stapedius muscles, acting both jointly and individually. The reflex action was elicited by tonal stimulation of the opposite ear. For excitation a little above the reflex threshold, the most striking effect is a reduction in the transmission of low tones. This effect diminishes as the frequency is raised and disappears around 600 cycles. It then becomes an enhancement for a narrow region between 600 and 1,000 cycles. For the higher frequencies, the tensor tympani muscle produces a moderate reduction in transmission, whereas the stapedius muscle has only a slight effect. When the acoustic stimulation is made stronger, the muscle action becomes more vigorous and the transmission undergoes progressively greater changes. For the low tones, the reductions of transmission keep pace with the stimulation for a considerable range of intensity and so there is a check on the magnitude of stimulation reaching the inner ear. The sensory structures of the cochlea are thus protected against damage, until finally a limit of the muscular action is reached. In some ears the regulatory effect may be as great as 20 db. for the lower tones.

The effects of succinyldicholine (I), decamethonium (II), and curare (III) on the middle ear muscles were investigated by Wersall. The stapedius and the tensor tympani are both blocked by III. The tensor tympani is also paralyzed by I and II, but these drugs cause a tonic contraction of the stapedius.

Laskiewicz discusses the innervation of the external auditory meatus and tympanic

membrane, the physiology of the Bechterew cochieofacial, and the Kisch auropalpebral reflexes. These reflexes are classified into separate categories, in the author's opinion. In 86 cases of middle ear and eighth nerve lesions, the relation between the sensitivity of the meatal wall and tympanic membrane and the reactivity of the vestibular apparatus could be established by Kisch's facial spasm and head tilting. In chronic catarrhal otitis media and otosalpingitis with ossicular stiffening, the auropalpebral reflex of Kisch becomes evident after Eustachian catheterization or bougienage. In post-traumatic, occupational, and detonation deafness, the auropalpebral reflex was feeble, but by use of the Galton whistle, Bárány's box, or Struyken's Monochord, the cochleofacial reflex of Bechterew appeared as the clearer reflex. In Ménière's disease and nerve deafness, the parallelism between the facility of elicitation of both reflexes and the recruitment phenomenon becomes evident. However, in hysteria and early otosclerosis, the separation of the two reflexes could be observed, the reflex of Bechterew being very marked; in late otosclerosis, both reflexes were weak. In toxic inner ear deafness, similarly both reflexes were only faintly present.

Vestibular Physiology

The occupants of a satellite, according to Lansberg, will have to face dangers and inconveniences, many of which have been dealt with in the pertinent literature. As such the state of weightlessness has been mentioned. A solution to this difficulty was found in the rotation of the satellite around its own axis, thus giving the occupants weight once again, though of a kind different from gravitational weight. The outer wall of the satellite will be the centripetal restraining agent to the centrifugal inertia force. This would be all very well if it were not for the angular velocity. The physiological consequence of this will be that any head movement around an axis not parallel to the axis of the satellite will engender untoward angular accelerations and deceleration of the endolymph, because the moment of inertia of the semicircular canals relative to the axis of the satellite will change concurrently with the head movements. False reactions and impressions are consequently elicited. The angular velocity mentioned in von Braun's concept of a satellite is 2/7 radial per second. Though trivial in the instance of a single impulse, this value might well prove to be beyond the limit of optimal physiological conditions for a prolonged sojourn during which man will need to give full scope to his faculties of handling, moving, acting, etc.

McNally gives his views on the function of the utricle. It responds to all kinds of linear acceleration, i. e., to gravitational acceleration, centrifugal acceleration, and acceleration accompanying linear displacements. The best-controlled experiments in which stimulation or ablation was strictly confined to the utricle have not elicited eye nystagmus. There is as yet no indication that such phenomena as directional preponderance or positional nystagmus result from stimulation or disease of the utricle. According to the author, the utricle has been shown to exert the dominant influence over labyrinthine tonic reflexes, righting reflexes, and compensatory eye deviations.

In considering and investigating labyrinthine function, Lansberg has expressed his findings under several headings: (*a*) A test person seated on a swing is asked to indicate sensations experienced. This is done to measure the threshold sensitivity of otoliths. The computed values were in the order of 6 cm. per second, which corresponds to an angular deviation of 1/30. After a series of experiments, doubt still existed about the otolithic origin of sensations experienced, particularly on differences noted when skin and deep sensations were absent or present. The parallel swing was also used to investigate counter-rolling movements of the eyes around a transversal axis. These movements could

not furnish any reliable indication of otolithic stimulation. (*b*) Steinhausen's concept of the semicircular canal is that of an aperiodic damped harmonic oscillator. This implies that the directive force of the cupula is a linear function of its deflection. The limit of tolerance to stimulation is dealt with, but the experimental results were inconclusive. (*c*) Caloric tests with critical temperatures are suggested as desirable in the examination of flying personnel. The tests are helpful in tracing disorders of the C. N. S., especially when we accept without dispute that the vestibular sense organ is involved in the origin of spatial delusions and motion sickness. (*d*) When the influence of vestibular stimulation upon EEG, ECG, and respiration was studied, the experiments left no doubt that the effects were of central origin. Vestibular stimulation, with the exception of optokinetic and tactile factors, changed the pattern of EEG, ECG, and respiration curve concurrently with the behavior of the nystagmus. (*e*) The tests based mainly on rotation were elaborated as a means of predicting a candidate's ability to pass the training and become a pilot. The findings, according to the author, seem to indicate a correlation between poor performance in the vestibular Adroitness Test and failure in the early stage of pilot training. (*f*) In an extensive and thorough study, Payne investigated the influence of anti-motion-sickness drugs. The author suggests that those persons predisposed to air sickness should be indoctrinated in dealing with vestibular or equilibrial impulses and that the aid of drugs should be used to break the chain in a vicious circle. The ban of drugs in trained pilots should on no account be lifted.

After injection of Chinese ink into the endolymph space, the cupula ampullaris can be visualized. A pressure of 0.1 mg. of water gives a deflection of the cupula equal to those seen during caloric tests. New facts are produced by Dohlman supporting a theory explaining the mechanism of the "fistular symptom" of the labyrinth. Here the hydrostatic pressure seems to be of no importance for the reaction. A certain deformation of the membranous labyrinth between the fistula and the round and oval windows seems to be essential.

After a review of the literature on the perilymph and the endolymph and on the embryology of the endolymphatic sac, four series of experiments with trypan blue injections are described by Brinkman. 1. Subcutaneous injection of 0.5% trypan (10 ml. per kilogram of body weight) into guinea pigs. From the connective tissue of the muscles and the fat tissue, the blue granules absorbed by histiocytes penetrate through the Haversian canals, along the blood vessels, into the labyrinthine capsule. The blue granules never penetrate into the scala tympani, the scala vestibuli, or the cochlear duct. In the endolymphatic space, blue granules are only found in the endolymphatic duct and sac (especially in the pars rugosa) and in the wall as well as in the cells of the lumen. Consequently, the uptake of trypan blue by the endolymphatic sac takes place from without and not by absorption of trypan blue from the rest of the endolymphatic space. 2. After destruction of the labyrinth, in which the endolymphatic sac was preserved, the cells of the epithelial wall and of the lumen of the endolymphatic sac were also found to be blue. 3. After injection of a trypan solution into the C. S. F., blue granules were observed in the cochlear aqueduct, the scala tympani, and the scala vestibuli. The entire endolymphatic space, including the endolymphatic sac, was completely free of trypan granules. 4. Trypan blue and streptomycin (300 mg. of streptomycin per kilogram of body weight) were injected simultaneously into guinea pigs. No differences from the experiments in Group I were observed. Nearly all of the microscopic preparations of the endolymphatic sac showed colorless globules, lying against the cells of the saccular wall; these were probably drops of secretion. It is concluded that the endolymphatic sac has a limited protective function for the rest of the endo-

lymphatic space and possibly also a secretory function.

The stimulation of the pigeon's ampullae of the semicircular canals by short pressure sounds (clicks) has permitted van Eyck to put in evidence an intermediary electrical potential, between the mechanical stimulus and the excitation of the vestibular nerve. The close parallelism which exists between this local electrical excitation and the variable sensibility of the semicircular canals, conformable to the laws of Ewald, pleads in favor of the essentiality of its role in the stimulation of the nerve. It seems likely that, provided the stimulus consisted of rotation instead of a click, the intermediary potential would manifest itself as a continued potential during the cupular deflection. Attempts to put in evidence such a continued potential have unfortunately been in vain because of insurmountable technical difficulties.

The permeability of the barrier between blood and labyrinthine fluids was studied by Buonsanto, Pinto, and Pennetta, who injected intravenously in the pigeon a solution of 1% radium bromide. This substance can be observed in the labyrinth and reaches its maximum concentration there after 30 seconds. A tendency to decrease appears after 45 minutes. In all experiments the radioactive solution concentration in the labyrinth and in other organs studied has been higher than that in the blood.

The vestibular compensation in the rabbit, following a transtympanum unilateral injection of alcohol at 60 C, appears late and at a level of improved bilateral function under the action of ethyl urethan administered intraperitoneally. It is difficult to derive any founded hypothesis from the fact that the compensation level appears to be higher after a labyrinthine lesion made in narcosis; for this purpose a hypothesis of work can be suggested, i. e., that the state of narcosis should prevent or attenuate the possible establishing of a central inhibition, perhaps a cortical one, during the violent crisis of vestibular derangement remarked in a nonnarcotized animal after a labyrinthine lesion. Beretta and Livan sum it up as follows: (*a*) The distinct and manifold action of urethan on the vestibular reflectivity and in particular on the compensation phenomenon suggests that such an effect should not happen only in the cortical part but should involve also those regions in the encephalon that interfere with the fulfillment of vestibular reflexes. (*b*) The compensation does not appear under hypnosis during all the time when the effect of the drug on the cortex is very evident. (*c*) The higher level where compensation appears bears a consequent improvement in the vestibular activity, which fact was already well known, in the clinic, of labyrinthine fenestration. Actually when the intervention takes place in general anesthesia, vertigo is not the usual predominant sensation during the postoperative course.

The effect of ethyl urethan shows off in two different ways, either by delaying the compensation itself or by allowing a much higher compensation level. It was also concluded that this drug, in addition to its well-known depressive effect on the cortical activity, exerted a peculiar effect on the vestibular activity itself. A problem should logically arise whether the dual effect corresponds in some ways to the dual action, i. e., whether the delay obtained was or was not connected to the cortical action of the drug, together with the raising of the functional level. With the purpose of solving the problem and of setting down in particular whether the considered compensation should be cortical or not, Beretta and Livan wanted to examine the behavior of the postrotational vestibular reflectivity in the thalamic rabbit after a one-sided transtympanum injection of ethyl alcohol. The removal of the cerebral cortex in the rabbit delays but does not prevent the forming of a vestibular compensation after a transtympanum injection of alcohol at 60 C. What has been studied permits them to consider as sufficiently proved the idea that the cerebral cortex, though facilitating compensation by an inhibiting action on the two vestibular apparatus, is not the

very seat of the phenomenon and is not indispensable to its happening.

Branca and Germana studied the possibility of localizing anaphylactic shock to a nonacoustic labyrinth in 15 guinea pigs by means of intensive sound stimulation. The rotation test was carried out with the animal intact, first before and again after application of the sound stimulus (maximum intensity of a tone of frequency 4,000 applied to the left ear for five minutes). Twenty minutes after the sensitization of the animals, the sound stimulus as described was repeated; then the shock-causing injection was given and the rotation test done again. No significant differences between the duration of nystagmus during the three tests could be found.

In 20 frogs, the left horizontal semicircular canal was eliminated by section of a branch of the vestibular nerve and cauterization of the ampulla. Shortly after the operation the animals, under different ways of stimulation, showed a tendency to turn to the side operated on, reports Gribenski. During examination on a turntable rotational reactions to the right were completely abolished. In two to three months after the operation, definite but weak reactions to the right reappeared. Rotational tests performed on six frogs, remaining alive after eight months, showed a certain degree of compensation. During turning to the left or after turning to the right was stopped, some reactions to the right could be observed. Nevertheless, they were weaker and less regular than the reactions to the left; also some tendency to turn to the side operated on still persisted. Elimination of the remaining (right) horizontal caused all the rotational reactions in the horizontal plane to disappear. Thus, it has been proved that in frogs, too, rotational reactions in both directions can arise from one horizontal canal. Of course, it does not necessarily follow that in a normal animal both horizontal canals actually participate in reactions to both sides. Experiments on a greater number of animals and prolonged observations are necessary to decide whether complete compensation can be established in the frog. In this case, there would be no tendency to turn to the side operated on and the rotational reactions to both directions would have the same strength.

The vestibular nerve of the frog, which divides directly from its point of origin, permits of separate study of the branches which innervate the various semicircular canals, the sacculus proprius, and the utriculus vestibuli. By separate registration of the action currents of these different nerve branches, it proved possible to study the function of the various elements of the vestibular apparatus. This method was then applied by Wallancien to the study of the effects of various toxic substances (cocaine, Arsacetin, Dramamine, streptomycin) on the various vestibular elements.

The significance of perilymph in stimulation of the semicircular canals is pointed out by Wustrow. Microscopic slices of human semicircular canals show that the diameter of the perilymphatic space is three to four times larger than the lumen of the semicircular canal. In the region of the ampulla this canal becomes so large that the perilymphatic space is nearly blocked. The ampulla here closes the osseous semicircular canal almost completely shortly before its entrance in the vestibule. It is suspended at the osseous wall by a narrow mesh of tissue, while in the region of the peripheral semicircular canal only a few fibers exist. These anatomic facts are of importance in the shifting of the labyrinthic fluid. Both endolymph and perilymph move. The mass of perilymph is three times greater than the mass of endolymph. In centripetal movements the perilymph exerts a marked pressure on the ampulla and deforms it, causing a narrowing of the space anterior to the cupula. The deformation of the cupula is further increased by the pressure of the endolymph. The deviation of the cupula is greater on the ampullopetal side than on the ampullofugal. This may explain the fact that in turning experiments nystagmus is induced by that labyrinth with ampullopetal move-

ments of fluids. Viscosity of peri- and endolymph and the external and internal friction are important. In caloric experiments cold water rinsing with an ampullopetal shifting of fluid is a greater irritation than hot water rinsing with the ampullofugal shifting.

In order to investigate the progressive appearance of compensatory phenomena in patients having only one labyrinth, 62 subjects who had undergone a unilateral labyrinthectomy for Ménière's syndrome were studied by Pivotti at different intervals after the intervention. Special attention was given to Ruttin's phenomenon, which bears the following characteristics: In a patient with one labyrinth, it can be noticed at various intervals after the operation that, while the caloric stimulation on the ear operated on fails to give a reflex of labyrinthine origin, the rotatory stimulation in both directions obtains in the first phase of the postrotatory nystagmus a symmetric reflex, as if the labyrinth was still able to elicit a vestibule response though not responsive to caloric stimulation. By use of different physical stimuli on subjects which had been operated on previously at various periods before the test, it has been observed that Ruttin's phenomenon is related to the intensity of the stimulus and to the time elapsed after the operation of labyrinthectomy. The author thinks that the phenomenon might be due not only to modifications arising in the peripheral organ, after a certain period of time has elapsed after the intervention, but also to central supranuclear compensatory mechanisms. Most probably both factors act at the same time.

A partial chemical lesion of the right labyrinth in rabbits, already submitted to the same lesion of the right labyrinth six days before, does not cause any variation in the vestibular postrotational reflectivity. According to Beretta and Livan, this can be considered very interesting, as the effect of a partial labyrinthine lesion appears to be very different on the vestibular reflectivity of a sound animal from that on an animal previously submitted to the same lesion in the other labyrinth and in compensation phase. Another interesting point concerns the vestibular crisis that shows up following the second transtympanum injection of alcohol. This crisis shows a peculiar feature in respect to the one caused by the first injection. In the first case, a short period of latency was followed by a labyrinth-lacking symptomatology that lasted during the whole time of the crisis; in the second case, after the same period of latency, a definite irritative symptomatology showed off, lasting about 20 seconds. This period was followed by a paralytic period, much less intense and lasting longer than the one following the first injection. This happens very clearly in all animals, with no exception. A wide field of hypothesis is opened after these remarks. According to the most fascinating ones, one would suppose that the damage provoked in the second labyrinth makes the compensatory central inhibition in vestibular apparatus unnecessary following the previous counterside lesion and would cause its regression. This might justify the absence of any remarkable variation in the vestibular reflectivity after the second injection.

Giulio examined in vivo (guinea pigs) the effect of ampullar excitement of the semicircular lateral canal given by endolymphatic currents caused by localized and instantaneous modifications of temperature corresponding to the ampullar region. The canal itself was oriented horizontally during the test. The author previously described the technique for the myographic optic registration of the phasic activity of the external rectus muscle contralateral to the stimulated labyrinth: slow contraction and quick relax, the reckoning of the contraction speed of the muscle and the thermoelectrical drawing of the local temperature. The thermic threshold for the minimum phase activity of the external rectus muscle is 2.14 C. A rise in temperature of little more than two degrees suddenly realized on the ampullar region of

the lateral canal when parallel to the horizontal plane is sufficient to cause a very low contraction of the external rectus muscle, followed by a quick relax. Under these conditions the muscle shows a single jerk for about five seconds. The threshold has a value over seven times the one obtained under similar conditions, but with a lateral canal inclined at 30 degrees with ampulla up (threshold, 0.3 C). By raising the intensity of the thermic stimulation in a horizontal canal, the speed of "low" contraction of the examined muscle rises too, together with the entity of shortening and the frequency of nystagmus. A purely physical hypothesis of nonexcitability in the ampullar receptor for impossibility of formation in the convective currents in the case of nonhorizontal canal proves to be not very feasible in vivo (nor in the "bridge" preparation) because of the secondary curves of the semicircular canal itself. The threshold after minimum physical activity of the external rectus muscle is much higher when the canal plane is horizontal, compared with the values obtained with an inclined canal. This fact corresponds to the previously stated physical deductions, as it proves that effective convective currents appear after receptor stimulation when the canal leaves its horizontal position.

The effects of galvanic stimulation of the labyrinth on the activity of a single unit of Deiters nucleus were studied by de Vito, Brusa, and Arduini, who point out the following results of their inquiries: (*a*) The anode polarization can be followed by a rise in the unitarian ipsilateral action, and so it should not possibly work in a constant way, blocking thus the spontaneous charge of the receptors or of the primary neuron. (*b*) The continuous presence of effects on the Deiters counterlateral nucleus shows the existence of several important crossed connections between the two vestibular systems. They do not in any way cross the cerebellum, as an acute total cerebellotomy would neither reduce nor abolish them. (*c*) The intensity threshold for the disappearing of variations in the unitarian Deiters discharge corresponds to the appearance of a threshold of oculocephalogyric movements, while the tonic variations in the muscles of the limbs appear with a higher intensity. This remark raises the problem of the behavior of the ampullar and otolith system, compared with the galvanic stimulation, and of their relation to Deiters discharges registered with threshold stimulation.

Labyrinthine reaction can be modified by unilateral lesions or stimulations of the cerebral cortex. These facts were usually regarded as dependent upon alterations of the vestibular nucleus, but, according to di Giorgio, even under these conditions the state of "common final way" should be kept present, as considered for the cerebellum. Actually the unilateral modification of some determined cortical impulses (excitement and inhibition) that reach the motor neuron interested in vestibular reactions may influence their character, without the necessary intervention of the vestibular nucleus. The above is confirmed by an inquiry on the cortical nystagmus area, which was discovered in guinea pigs some time before. The author proved that the "common final way" (ocular motor neurons) is subject to a series of cortical impulses which are not surpassed by the ones caused by labyrinthine excitement. On the other hand, when one labyrinth is suppressed, the excitement of the contralateral cortical area will either give no reaction for several days or give an anomalous or inverted response to the regular one. In this case, the state of the ocular motor neurons is modified by the asymmetrical labyrinthine impulse and the cortical excitement will not have any effect until the moment when such asymmetry results are more or less compensated. The contemporary destruction of the two labyrinths permits the regular evincing of the cortical nystagmus a short time after operation, being the ocular motor neurons of the two sides in similar conditions compared with the extralabyrinthine excitement. As no relation between the

cortical-fugal way in the considered cortical zone and the area of the vestibular nucleus is foreseen, it can obviously be concluded that a clear interference exists at the level of the "common final way." The same factors would be at the base of the vestibular alterations in case of an asymmetric cerebellar action, as well as in the case of asymmetric cortical activity. The state of the common final way might be modified by a single group of exclusively cortical impulses and not by others derived from nervous formations secondarily interested, as probably happens in the cerebellar lesion. It can thus be explained that the anomalies in the labyrinthine reaction in case of modified cortical activity are of limited importance and relatively transitory as compared with the anomalies of cerebellar origin.

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Abstracts from Current Literature

Ear

THE FENESTRATION OPERATION: A PERSPECTIVE IN TIME. ROBERT C. BIGLER, ALLAN C. GOODMAN, and T. WALSH, Laryngoscope 69:141 (Feb.) 1959.

The report is a continuation of a long-term study of the results of tests (spondaic words) of hearing on fenestrated patients. It deals with the first 818 patients operated upon. These are divided into three chronological groups, and the results compared.

Many aspects are discussed. They include hearing gain at various times postoperatively, closures, stability of hearing, and the threshold and interpretation of a mathematical analysis of these data. The paper must be studied carefully to appreciate its full worth.

HITSCHLER, Philadelphia.

LOUDNESS RECRUITMENT AND VERTIGO. WALTER ALEXANDER, Laryngoscope 69:174 (Feb.) 1959.

The binaural loudness balance test is described. It is usually performed at 1,000 db. The threshold values of each ear are recorded and the sound intensities necessary above threshold for the loudness to seem equal in both ears. In an ear with recruitment, sounds well above threshold are heard as loudly as those in the normal ear. In other words, as the signal is raised above threshold, there is a greater gain in its perception in the diseased than in the normal ear. Later, another test, the difference limen test, was proposed. This is a test of the patient's ability to detect a small change in the loudness of the signal. In an ear with recruitment, a smaller difference in loudness can be detected than in a normal ear. Several variations of this test have been used. These tests have the advantage of being able to be used on monaural hearing losses. Many have verified their usefulness in indicating recruitment.

Recruitment occurs in perceptive deafness. Histological studies on the cochlea and eighth nerve have been performed that indicate a retrocochlear lesion does not affect the cochlea unless it interferes with its blood supply. Ménière's disease and acoustic trauma do affect the cochlea. Investigation of cases of retrocochlear lesions show that they in general do not show the recruitment phenomena, while those of the cochlea itself do.

The physiological and anatomical concepts of recruitment are discussed. In a damaged cochlea there are fewer hair cells. Weak stimulation gives little cochleal stimulation. However, with strong stimulation these fewer hair cells do become well stimulated and are able to convey impulses of normal intensity to the cerebral cortex.

Another phenomenan noted with recruitment is the less of speech intelligibility as loudness increases.

HITSCHLER, Philadelphia.

ENDOLYMPHATIC CIRCULATION OF THE COCHLEA. ETTORE BORGHESAN, Acta oto-rino-laring. ibero.-am. 10:126, 1959.

The author contends that the tectorial membrane is united to the ciliated cells and represents the elongated cilia of these cells. Accordingly, the cochlear duct is divided into two large endolymphatic spaces—one is the true cochlear duct, and the other is the spinal canal.

According to the author, the vascular stria secrete the endolymph. This circulates in a radial direction from without inward, passes through the duct formed between the organ of Corti and the tectorial membrane, and is absorbed by the limbus.

PERSKY, Philadelphia.

LAXITY OF THE OSSICULAR ARTICULATIONS IN OTOSCLEROSIS AND ADHESIVE OTITIS. R. MASPÉTIOL, MATHIEW, and TRONCHE, Ann. oto-laryng. 75:844 (Dec.) 1958.

The authors describe a rare syndrome of deafness. This may be due to isolated lesions of the articulations of the ossicles or may be associated with an ankylosis of the stapes. In the former instance, there may be a relaxation of the articulation of the incudostapedial joint or the incudomalleolar joint or both. Audiometrically, there is a deafness of transmission with a definite trough at the 1,000 frequency level. The Gelle test is positive but inversed.

In the latter group, one finds an adhesivable otitis rather than a true otosclerosis. The relaxation of these various articulations may be the reason for some of the failures that are

encountered in cases which ordinarily would improve with a simple mobilization operation. The authors could not predict any clinical evidence that can be established with this diagnosis preoperatively, but in a few cases they were able to note this condition at the time of operation.

PERSKY, Philadelphia.

ARTERIOVENOUS ANEURYSMS OF THE EAR AURICLE, THE GLOMERULAR TYPE. R. GRIMAUD and M. WAYOFF, Ann. oto-laryng. 75:875 (Dec.) 1958.

The authors present two rare and interesting cases of cirsoid aneurysm in the region of the ear. The first involved the anterior and posterior aspects of the auricle, with extensions in front of the ear and also inferiorly and posteriorly. At the time of operation the internal carotid artery was ligated and an ampoule of 25% of Sclerona solution was injected into the anterior and posterior sacs of the aneurysm. The anterior sac disappeared completely, but the posterior sac remained. Two months later the posterior sac was resected, and the patient made a very satisfactory recovery.

The second case was that of an arteriovenous aneurysm in the retroauricular region. This was diagnosed by an arteriogram. Hereto, an operation was performed which entailed the ligation of the external carotid artery and then the sac was completely dissected. Although a considerable amount of bleeding was encountered, this was controlled with additional ligations. After the operation, the patient made a very uneventful recovery.

PERSKY, Philadelphia.

MOBILIZATION OF THE STAPES. R. MASPETIOL, MATIEU, and SEMETTE, Ann. oto-laryng. 76:27 (Jan.-Feb.) 1959.

The authors discuss many of the problems encountered in the surgical technic of mobilization of the stapes. They employ the postauricular approach. After exposing and elevating the tympanic membrane, they use a burr to reduce the posterior superior angle of the external canal. This will bring into view the stapedius muscle, the posterior crura, and the footplate. They then free the footplate by making linear incisions along the periphery parallel to the annular ligament. They begin the mobilization at the posterior pole and circle the area where the posterior crus is attached. Moving forward the bistoury is inserted into the margin of the footplate and the oval window, at the base of the anterior crura, below and in front of the crus.

The immediate postoperative results are excellent. However, there are a number of secondary developments that may influence the ultimate result. These are tubal catarrh, edematous reaction of the mucous membrane of the tympanic cavity, unsuspected fracture of the crura, secondary fibrosis of the footplate due to laceration of the mucous membrane, and injury to the cochlea.

In their series, the authors had very few operative complications. These include fracture of the crura, a transverse fissure of the footplate with a loss of a portion of the footplate. Permanent facial paralysis was not encountered, although there were a few cases of a transient paralysis due to the anesthesia. Tearing of the tympanic membrane occurred in a few cases. Vertigo occurred in a few cases but lasted only 24-48 hours.

In conclusion, the authors believe that the direct mobilization of the footplate is the operation of choice, and have obtained a functional success in 80% of their cases. This implies an auditory gain of at least 30 db. Injury to the cochlea was not observed, and vertigo was rare. They prefer this technique to that of indirect mobilization or fenestration of the footplate.

PERSKY, Philadelphia.

AUDITORY PERCEPTION IN CHRONIC SUPPURATIVE OTITIS MEDIA WITHOUT CHOLESTEATOMA. F. GUNNEL, Arch. Ohren- Nasen- u. Kehlkopfh. 174:150, 1959.

Ever since the introduction and development of the tympanoplasty operation, the indications and methods of surgery for chronic suppurative otitis media have undergone a complete revision. Of prime importance is the role that conductive deafness plays in establishing newer criteria for surgical indications. In consequence of these problems, the author has attempted a study to determine the relationship of loss and anatomical findings in cases of chronic suppurative otitis media. A number of questions must be answered. 1. Is there a difference in the hearing loss in cases of chronic otitis media with a typical central perforation against those cases with a marginal perforation? 2. Does the difference in the size of the perforation in the membrane tensa have any influence on the hearing loss? 3. Can one predict how great the hearing loss is in suppuration of the mucous membrane? Does this extend to the limit

of interfering with the social activities? 4. Is the once established hearing loss stationary, or will it become progressively worse, and, if so, in how long a period of time? 5. What is the relationship to the bone-conduction curve?

There were three groups of patients that the author has classified. 1. A chronic infection of the mucous membrane with a central perforation of tympanic membrane. In this group there were 510 patients. 2. Chronic otitis media with a marginal perforation in the membrana tensa but an intact Shrappnell's membrane. There were 291 patients in this group. 3. Chronic otitis media with a subtotal or total defect in the tympanic membrane but without the presence of cholesteatoma. There were 241 patients in this group.

Comparisons were made in the following groups: 1. A pure mucous membrane suppuration compared with a chronic otitis media with a marginal perforation. 2. Mucous membrane suppuration compared with a chronic otitis media with subtotal or total defect in the tympanic membrane. 3. A comparison of No. 1 and No. 2 in the various age groups. The author has made the following deductions: 1. There is no significant difference in the hearing loss between a suppuration with a central perforation and one with a marginal perforation. Both groups have an equal degree of impairment of hearing. 2. There is a marked difference in the hearing loss between the first group and that where there is a subtotal or total destruction of the tympanic membrane. Invariably, the hearing loss is much greater in the latter group. 3. Finally, there is not a great deal of significance between the hearing loss at the various age groups. What applies to one age group is similar to the next age group. In general, the author found that hearing loss does not remain stationary but becomes progressively worse over a period of time.

PERSKY, Philadelphia.

THE TREATMENT OF VERTIGO AND TINNITUS IN ATHEROSCLEROSIS. J. CALVET and A. RIBET, J. franç. oto-rhin-laryng. 7:585 (July-Aug.) 1958.

Atherosclerosis is one of the most frequent causes cochlear syndromes (deafness of the perceptive type and tinnitus) and certain cochlear labyrinthine syndromes, as evidenced by labyrinthine vertigo.

In many of these cases presenting these symptoms—perceptive deafness, tinnitus, and vertigo—the authors have found a high cholesteremia, particularly, the total cholesterol, β-lipoproteins, and the total lipids. They treated a series of 23 patients with "Solvosterol," which is composed of five amino acids isolated from a portion of the molecule of protamide. Five intramuscular injections of 5 cc. of a 2% solution are given within a period of 15 days. Where the blood chemistry does not show a marked improvement, they supplement a sublingual tablet of this preparation. One tablet is given three times a day for 10 days each month. In this series of cases, the authors have found a marked improvement in the blood chemistry.

On the other hand, altho there was a slight improvement in the tinnitus and vertigo, these findings are equivocal. The reason for the disappointing results is that usually the atherosclerosis has reached an irreversible status. Stress is made on the importance of instituting these studies very early—as soon as the patient begins to complain of either one or all three symptoms. They believe that early initiation of treatment can cure many of these cases or at least retard the progression of the pathology so that it does not reach the irreversible state.

PERSKY, Philadelphia.

SEROUS OTITIS. RAUL VELASCO, Rev. otorrinolaryng. 17:14 (June) 1957.

The author believes that serous otitis is an independent clinical entity and has its own etiopathology. While the clinical picture may resemble that produced by other conditions, such as a tubotympanic catarrh, catarrhal otitis media, blockage of the eustachian orifice, changes in barometric pressure, and allergy, there is a group of cases that does not fall into any of the above categories.

In a series of 54 cases, the etiology is rather vague—mild or severe coriza, barothrauma, or unknown cause. The symptoms include a sensation of fullness in the ear, with or without a slight amount of pain, moderate impairment of hearing, occasionally a mild vertigo, and usually no fever. The otoscopic picture often is rather inconclusive. The audiogram in 18 cases showed a horizontal loss of about 30 db. in all frequencies, and in 36 cases there was a marked drop in the upper frequencies.

Allergic factors, found in 46% of the cases; neurovegetative disturbances; endocrine imbalance; pregnancy, and cardiorenal disturbances account for some of the other causes.

The most satisfactory method of treatment was puncture aspiration of the middle ear. This is preferable to paracentesis.

PERSKY, Philadelphia.

Larynx

EARLY DIAGNOSIS AND TREATMENT OF CORROSIVE BURNS OF THE ESOPHAGUS. EUGENE L. YURICH, Laryngoscope 69:131 (Feb.) 1959.

Early diagnosis is important. The history and appearance of burns about the mouth and lips, together with pain, dysphagia, and hoarseness, are indicative. With extensive burns shock and dyspnea due to laryngeal involvement, the diagnosis is obvious.

Roentgenograms and fluoroscopy are not considered worth while, but early (12-24 hours) esophagoscopy (under general anesthesia) is most important.

Treatment consists of first neutralizing the acid or alkalis with appropriate solutions. Yurich does not favor the use of gastric tubes because of their possible traumatic effect. Also early (12-24 hours) the patient is put on penicillin and the steroids, the latter on relatively large doses. Diet is liquid at first; then soft; then, in a week, solid food. Esophagoscopy is repeated every 10 days until healed. With this treatment 8 of 10 cases responded well.

When there is no diagnosis within 48 hours, the steroids cannot be used. Daily dilatations then have to be resorted to. Details of late treatment are given.

HITSCHLER, Philadelphia.

News and Comment

ANNOUNCEMENTS

Fifty-Seventh French Congress of Oto-Rhino-Laryngology.—The Fifty-Seventh French Congress of Oto-Rhino-Laryngology will take place from Monday, Oct. 19, to Thursday, Oct. 22, 1959, in the Grand Amphitheatre of the Faculty of Medicine, rue de l'Ecole de Medecine, under the presidency of Dr. A. Soulas, of Paris, with the honorary presidency of Prof. L. Binet, member of the Institute, Dean of the Faculty of Medicine of Paris.

Numerous papers will be presented, and two reports will be discussed:

1. Surgery for Deafness, Its Present State, Its Future.
   Reporters: Messrs. G. Portmann, M. Portmann, and G. Claverie.
2. The Laryngologist and Actual Data on Treatment of Acute Respiratory Insufficiencies.
   Reporters: Mr. Paul Aboulker, with the collaboration of J. Lissac and O. Saint-Paul.

An exposition of surgical instruments, appliances, and pharmaceutical specialties interesting to the otorhinolaryngologist will be held in the Hall of the Faculty.

For all information, write to the General Secretary, Dr. H. Guillon, 6 avenue Mac Mahon, Paris 17.

Books

BOOKS RECEIVED

* **Surgery of the Ear.** By George E. Shambaugh Jr., M.D. Price, $27.50. Pp. 669, with 326 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5, 1959.

Die Speicheldrüsen des Menschen: Anatomie, Physiologie und klinische Pathologie. By Sigurd Rauch, Dr. Med., Dr. Phil., and Dr. Eslettres. Price, $18.80. Pp. 507, with 227 illustrations. Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart N, Germany; Intercontinental Medical Book Corporation, 381 4th Ave., New York 16, 1959.

BOOK REVIEWS

Treatment of Cancer and Allied Diseases: Vol. 3, Tumors of the Head and Neck. Edited by George T. Pack, M.D., and Irving M. Ariel, M.D. Price, $3. Pp. 850, with 1,028 illustrations. Paul B. Hoeber, Inc. (medical book department of Harper & Brothers), 49 E. 33d St., New York 16, 1959.

This third volume, entitled "Tumors of the Head and Neck," is one of the most comprehensive and illustrative publications available on the subject. It is of particular interest to otolaryngologists, since it provides an over-all perspective of general anesthesia for head and neck surgery, local anesthesia, and pre- and postoperative care of surgery of the head and neck. Many subjects not generally included in books specifically for the otolaryngologist are completely covered in this publication. Among these subjects are lesions of the oral cavity, the lips, the buccal mucosa, the tongue, the floor of the mouth, the tonsil, and the pharynx. Comprehensive discussions of surgical and irradiation therapy are included. All phases of malignancies and their treatment are described and illustrated in this volume. There is a special section for tumors of the ear and larynx and of the neck, including carotid body and other lesions of the neck. This is an excellent publication and should be included as a reference in the library of every otolaryngologist.

Eye, Ear, Nose and Throat Manual for Nurses. Eighth edition. By Roy H. Parkinson, M.D., F.A.C.S. Price, $3.85. Pp. 237, with 82 (including 2 in color) illustrations. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1959.

This book gives an excellent source of basic factual information for the student nurse. Part 2 is especially excellent as a reference to the student gaining experience in the operating room, or for the postgraduate student specializing in operating room or ophthalmic nursing.

Current nursing education stresses total-patient care. The author discusses few or none of the emotional problems of chronic disorders of the eye, ear, nose, or throat. There is a need for an expansion of the psychological preparation of the patient for surgery, as well as the handling of the patient following surgery. In addition, there is a lack of community resources for patient referral, which should be known by the nurse to meet the rehabilitative needs of the patient.

The greatest drawback found was the absense of a bibliography or additional references, which could be referred to by both the instructor and student nurse.

* To be reviewed.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. G. Arnold Henry, 170 St. George St., Toronto.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Rd., Halifax, N. S.
Meeting: Sheraton-Brock Hotel, Niagara Falls, Ontario, Oct. 9-10, 1959.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. John R. Lindsay, 950 E. 58th St., Chicago.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.
Meeting: 24th Annual Congress, Palmer House, Chicago, Sept. 14-18, 1959.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Paul H. Holinger, Chicago.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Meeting: Deauville Hotel, Miami Beach, Fla., March 15-16, 1960.

American Laryngological Association

President: Dr. William J. McNally, Montreal.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960.

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.

American Rhinologic Society

President: Dr. Kenneth H. Hinderer, 402 Medical Arts Building, Pittsburgh 13.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Oct. 8-9, 1959, Illinois Masonic Hospital, Chicago.
Annual Meeting: Oct. 10, 1959, Belmont Hotel, Chicago.

American Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8.
Vice-President: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Meeting: March 13-14, 1960.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Raymond S. Rosedale, 610 N. Market Ave., Canton 2, Ohio.
Secretary-Treasurer: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.
Meeting: Oct. 11, 1959, Conrad Hilton Hotel, Chicago, and March 6-13, 1960, Deauville Hotel, Miami Beach, Fla.

American Society of Facial Plastic Surgery

President: Dr. Oscar J. Becker, Chicago.
Vice-President: Dr. Sam H. Sanders, Memphis.
Secretary: Dr. Samuel M. Bloom, 123 E. 83d St., New York 28.
Treasurer: Dr. Joseph C. Miceli, Brooklyn.
Meeting: Oct. 15, 1959, Chicago.

American Society of Ophthalmologic and Otolaryngologic Allergy

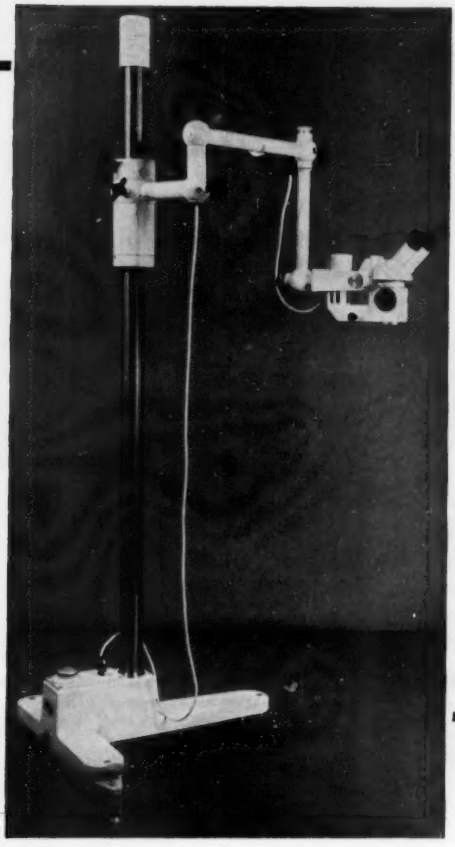
President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.

Otosclerosis Study Group

President: Dr. E. P. Fowler Jr., 180 Fort Washington Ave., New York 32.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2.

Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
Secretary-Treasurer: Capt. Maurice Schiff (MC), U. S. N., U. S. Naval Hospital, Oakland, Calif.



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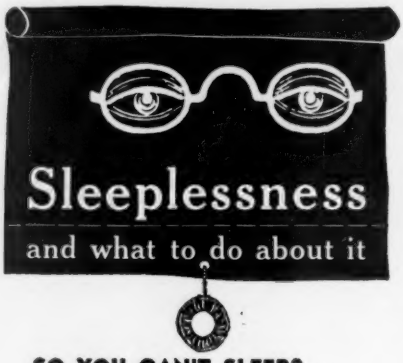

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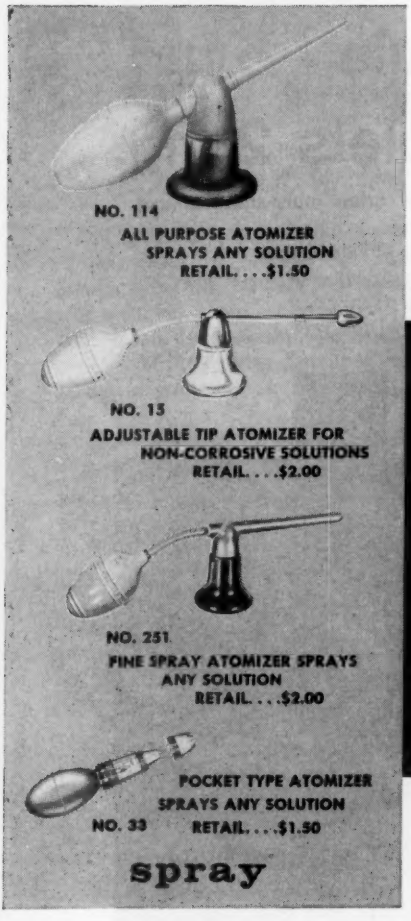

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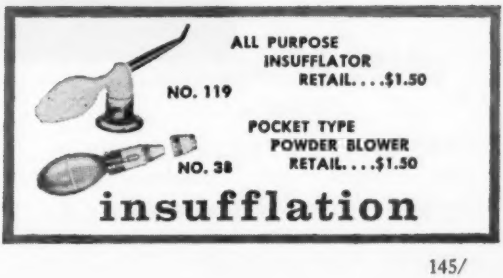

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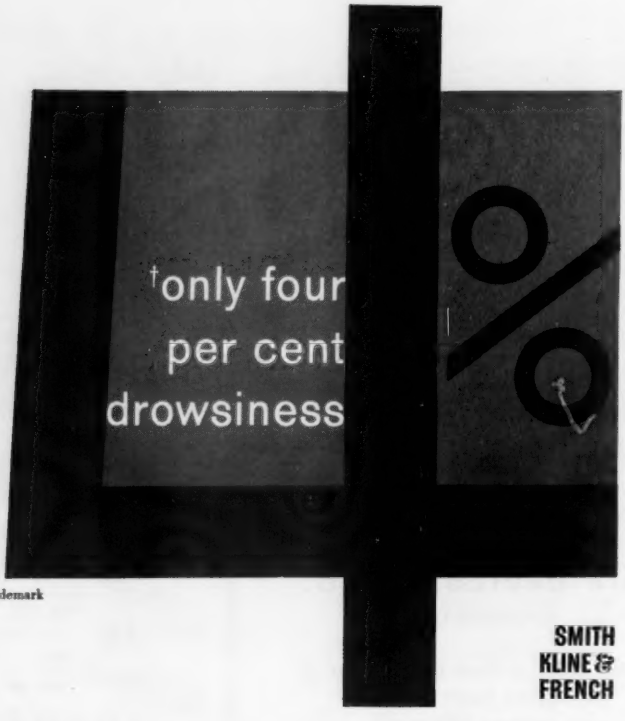

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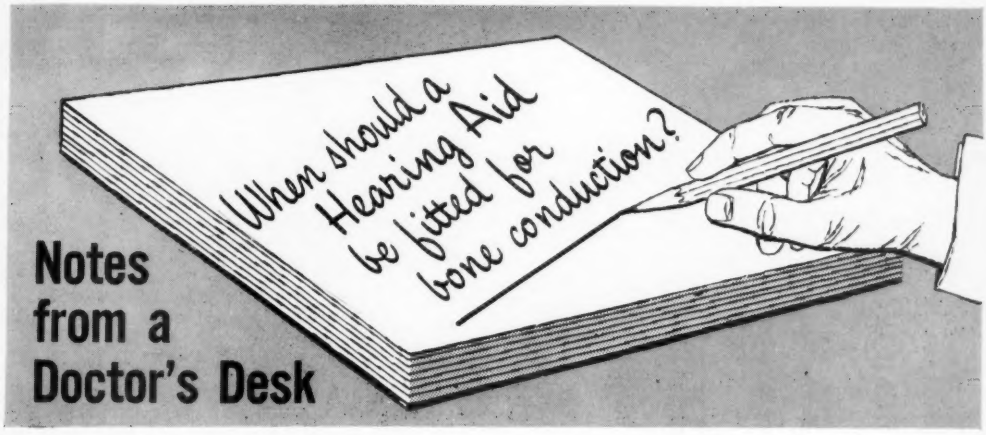

Notes from a Doctor's Desk

Questions Asked by Doctors About Some of Maico's Products:

Hardly a day passes without the newspapers or magazines carrying an ad announcing a hearing aid "with nothing in the ear." Generally speaking, these advertisements are for a bone conduction type aid, with the receiver concealed in an eyeglass temple or held in place by a headband.

Bone Conduction Requirements

Perhaps the most frequently made error in hearing aid recommendations is the prescription of a bone conduction fitting because of the presence of conductive deafness and moderately increased bone conduction.

According to generally accepted practice, bone conduction should be recommended over air conduction only:

—where a suppurative ear or other condition precludes the use of air conduction.

—where the patient's threshold for bone conduction is at least 20 db. better than for air conduction in the speech range tones (500 through 2,000 cycles).

Bone Conduction Fittings Under 1%

In the Audiology Centers established by the U. S. Army to handle hearing loss cases at the end of World War II, only 60 cases out of over 6,000 – less than one per cent – were fitted with bone conduction. They found that with a properly fitted earpiece, an aid with an air conduction receiver provided better discrimination and understanding. Furthermore, most hard of hearing persons prefer the comfort of an individually-molded earpiece to the headband arrangement necessary for good bone conduction.

MAICO Fits All Losses

The MAICO Dealer in your city has been thoroughly trained so as to understand the importance of correctly fitting a hearing aid to the specific requirements of the individual. With a complete line of precision-built hearing aids and hearing glasses, he is able to fit properly all types and degrees of hearing losses.

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Dealers in 275 cities in U. S. and Canada

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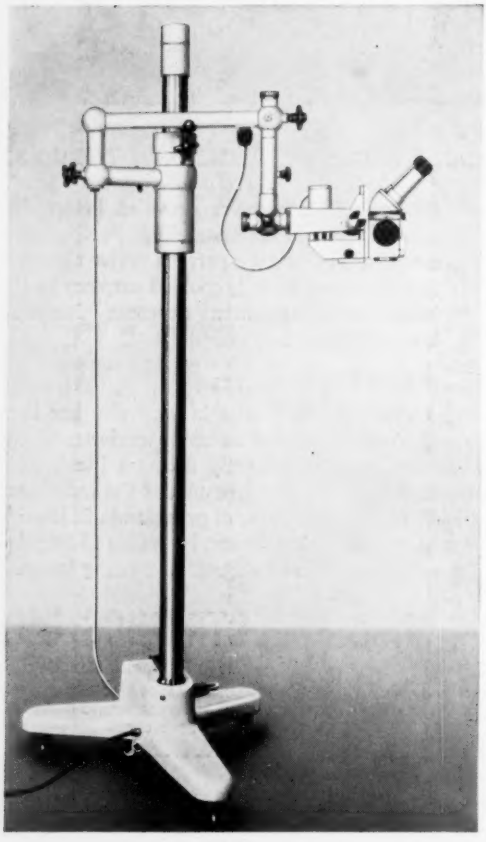

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